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BRAIN.

PART 1, VOL. 56.

THE DERMATOMES IN MAN.[1]

BY PROFESSOR O. FOERSTER,
Breslau.

The nervous system preserves more than any other part of the human body the original division of the embryonic tube into metameres. The spinal cord is divided in segments, each segment being provided with a pair of anterior and posterior roots. The area of skin which is supplied by the fibres of a certain spinal root is called a *dermatome*.

The skin is innervated by the posterior roots as well as by the anterior. The former carry the afferent sensory fibres and efferent fibres subserving vasodilatation. The anterior roots carry efferent motor, sudorific, pilomotor and vasoconstrictor fibres, and also afferent sensory fibres. In this lecture I shall deal exclusively with the fibres passing by the posterior roots.

There exist two different methods for the determination of the area of skin subserved by a certain posterior spinal root, the *anatomical method* and the *physiological method*.

The anatomical method, used first by Herringham and later chiefly by Bolk, consists in following up the fibres of one single root by anatomical dissection through the plexus and the peripheral nerves into the skin. Fig. 1 shows the second, third and fourth cervical dermatomes as outlined by Bolk by this method, and fig. 2 the areas of the roots supplying the arm, that is the fifth, sixth, seventh and eighth cervical and the first and second thoracic roots. Each of these dermatomes occupies a different area and these show very little or no overlap.

It is obvious that this method, used by Bolk, has the disadvantage that by anatomical dissection it is impossible to follow the terminal and

[1] The substance of this article was included in the Schorstein Lecture delivered at the London Hospital, on October 13, 1932.

finest ramifications of the sensory nerves in the skin. The areas outlined by it certainly show the shape and the position of the dermatomes, but not their full extent.

To Sir Charles Sherrington we owe our knowledge of the complete topography of the dermatomes in the monkey. The method used by him, and called the method of "remaining sensibility" or the "isolation method," consists in dividing a series of contiguous roots above and below a single root which is preserved. The area of the skin, the sensibility of which is preserved after this procedure, represents the sensory dermatome of the intact root.

FIG. 1.

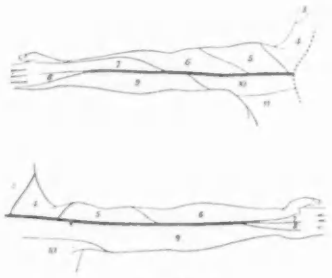


FIG. 2.

FIG. 1.—The 1st, 2nd and 3rd cervical dermatomes, according to Bolk.

FIG. 2.—Dermatomes of the upper extremity; the 4th to 8th cervical, and 1st and 2nd thoracic (9 and 10), according to Bolk.

Fig 3 demonstrates the second, third and fourth cervical dermatomes of the monkey; these overlap to such a degree that the area of the third cervical root is supplied to its full extent by the second and the fourth cervical roots.

A similar condition exists in the upper extremity (fig. 4). The fifth cervical dermatome, for instance, overlaps with the third and fourth on the one hand and with the sixth and the seventh on the other. The amount of the overlap of the different dermatomes is so considerable that division of a single root produces no loss of sensibility.

Another physiological method for the definition of the dermatomes is based upon local strychnine intoxication of the posterior roots. After segmental application of strychnine to the dorsal surface of the cord a sharply circumscribed skin field becomes hyperæsthesic, as Dusser de

Barenne has demonstrated. The hyperæsthetic area is identical in shape, situation and extent with the dermatomes defined by the isolation method, and the same large degree of overlap of adjacent dermatomes is apparent.

Our knowledge of the dermatomes in man is chiefly based upon the work of Sir Henry Head, who collected a large number of cases of herpetic eruption. Herpes is supposed to be an inflammatory lesion of

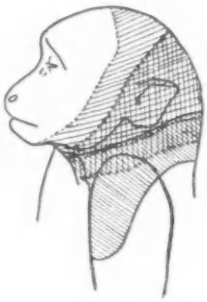


FIG. 3.

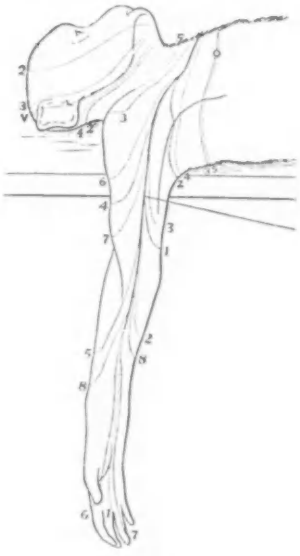


FIG. 4.

FIG. 3.—The 1st, 2nd and 3rd cervical dermatomes of the monkey, according to Sherrington.

FIG. 4.—Dermatomes of the upper extremity of the monkey, according to Sherrington.

the spinal ganglia, producing an eruption upon the skin in the corresponding cutaneous area. Head also contributed valuable information on the dermatomes in man by outlining carefully the borders of the hyperæsthetic skin areas in visceral disease, and the distribution of anæsthesia in traumatic lesions of the cord and the cauda equina. In Head's scheme (fig. 5) the cutaneous root areas overlap very little, and in this respect it does not conform to the results of experimental physiology. How is this discrepancy to be explained? Do monkeys and men differ really from each other so much in regard to such a fundamental organization as metamery?

I have had the opportunity of defining a great number of dermatomes in man by exactly the same method as that used by Sherrington, that is by outlining the borders of the sensibility which remains after a large number of contiguous roots have been divided, and a single root in the middle of them has been left intact. I need not discuss here the circumstances under which such a selected procedure may be undertaken. By this method I have been able to define all dermatomes of the lower

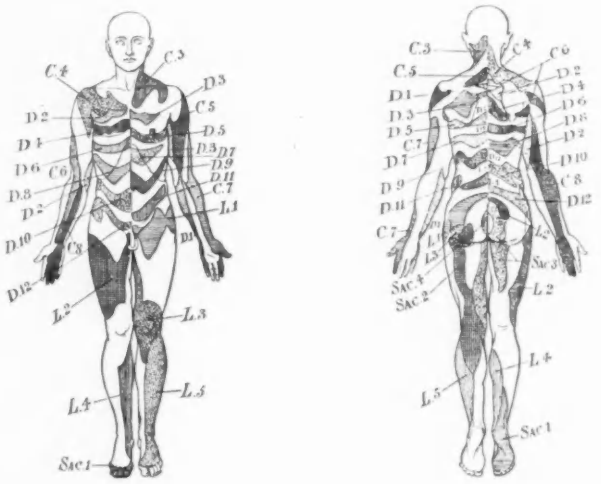


FIG. 5.—Head's scheme of the dermatomes in man.

extremity and one dermatome of the upper extremity, namely the sixth cervical. In order to fill up the gap and to define the other cervical and the thoracic dermatomes we can use a method which I should like to call "the constructive method." It is obvious that when a series of contiguous roots is divided, the superior border of the resulting anæsthesia represents the inferior border of the dermatome which corresponds to the next higher intact root, while the inferior border of the anæsthetic area represents the superior border of the next lower dermatome. By such observations I have been able to map out nearly all dermatomes in man.

The other method which I have used for the definition of dermatomes is based upon the fact, first demonstrated by Stricker and Bayliss on animals, that faradic stimulation of the distal part of a divided posterior

root is followed by vaso-dilatation. I have found the same in man. I have also observed that the vaso-dilatation following the electrical stimulation of a single posterior root is limited to a sharply circumscribed area similar in shape and situation to the dermatome as defined by means of the isolation method.

I have obtained vaso-dilatation by stimulation of nearly all cervical, thoracic, lumbar and sacral posterior roots. The area of vaso-dilatation resulting from the electrical stimulation of a single posterior root is not absolutely identical with the area of remaining sensibility; it is smaller and approximates in its extent much more to the area of herpetic eruption mapped out by Head.

For a long time it has been an open question whether the vaso-dilatation following faradic stimulation of a posterior root is due to antidromic conduction along the afferent nerve-fibres, as Bayliss believed, or whether it is the effect of stimulation of efferent fibres arising in the cord and passing outward by the posterior roots. In eight cases in which I had divided posterior roots in men we found a number of myelinated fibres preserved in the central part of the divided root. The root resection was performed in one of my cases five years before death (fig. 6), and in another four and a half years before death. The fact that these fibres are preserved after the root had been totally divided proves that their ganglion cells do not lie in the spinal ganglia but in the cord, and that these fibres are really efferent. The calibre of these fibres is very fine as compared with the thick myelinated sensory fibres of the posterior root (figs. 7 and 8.) The results of these anatomical investigations prove, I believe, that the posterior roots carry, in addition to sensory fibres, efferent fibres and it seems to me admissible to assume that the vaso-dilatation following the electrical stimulation of a posterior root is the result of the stimulation of these efferent fibres.

After this short review of the methods of defining the dermatomes in man I shall describe the special dermatomery. I begin with the dermatomes of the lower extremity.

Lumbar and Sacral Dermatomes.

First lumbar dermatome.—The first lumbar dermatome, that is the area of sensibility which remains after the eleventh and twelfth thoracic and the second to the fifth lumbar and all sacral roots having been divided, the first lumbar root alone being left intact, occupies the inferior part of the abdomen and the upper part of the anterior and lateral

FIG. 6.

FIG. 7.

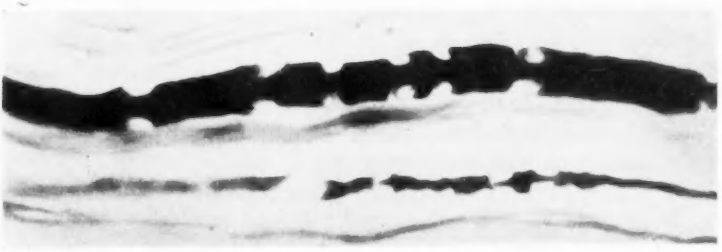


FIG. 8.

FIG. 6.—Efferent myelinated fibres of the central part of a divided posterior root, 4½ years after resection.

FIG. 7.—A single myelinated efferent fibre of a posterior root; note its fine calibre.

FIG. 8.—A single myelinated sensory fibre of a posterior root; note its thick calibre.

surfaces of the leg. At the dorsal side it forms a band, which reaches the spine of the fifth lumbar vertebra (figs. 9 and 10). This area of remaining sensibility is very similar to the area outlined by Head (fig. 5), but exceeds it to some extent, especially above its superior border. The extent of the herpetic eruption in a case of herpes of the first lumbar ganglion is shown in fig. 11.

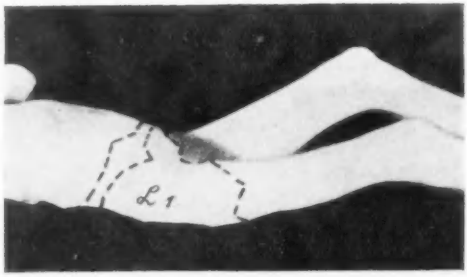


FIG. 9.

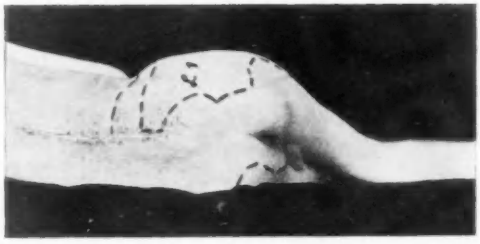


FIG. 10.

FIGS. 9 and 10.—The 1st lumbar dermatome defined by remaining sensibility after resection of 11th and 12th thoracic, and 2nd to 5th lumbar and 1st to 5th sacral roots.

The constructive method defines the same borders for the first lumbar dermatome. In a case in which the eighth, ninth, tenth, eleventh and twelfth thoracic roots were resected, the inferior border of the anæsthesia, shown in fig. 12, represents the superior border of the first lumbar dermatome; it conforms to the superior border of the remaining sensibility in the case described above. In another case, in which the second, third, fourth and fifth lumbar and all sacral roots were divided, the superior border of the anæsthesia shown in figs. 13 and 14

was practically identical with the inferior border of the remaining sensibility in the case illustrated in figs. 9, 10. In a case of division of the second and third lumbar roots the superior border of the anæsthesia was similar to that of the preceding cases on the anterior aspect of the leg, but on the posterior aspect there was no anæsthesia, the sacral roots having been left intact (figs. 15, 16).

Second lumbar dermatome was defined by the isolation method in a case in which the twelfth thoracic and the first lumbar, as well as the third, fourth and fifth lumbar and all sacral roots were divided (fig 17).

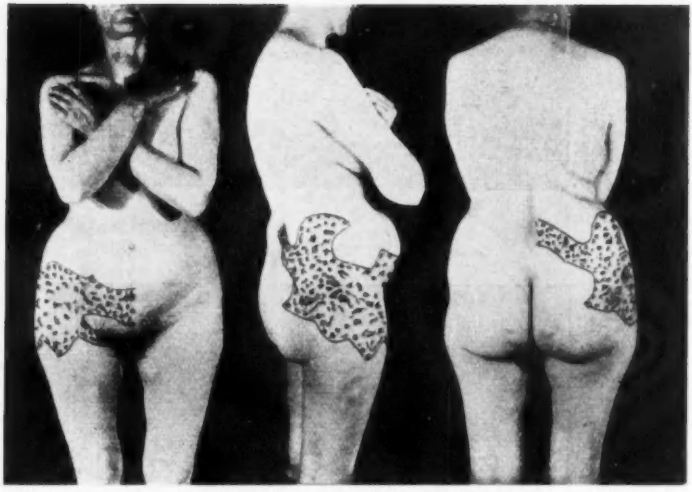


FIG. 11.—Herpes zoster of the 1st lumbar ganglion.

It lies chiefly on the anterior aspect of the thigh, but there is also a small band on the dorsum of the trunk which is not continuous with the main area upon the anterior surface of the leg (fig. 18). We find exactly the same distribution in Head's scheme (fig. 5).

The constructive method demonstrates the same borders for the second lumbar dermatome. In a case in which the third, fourth and fifth lumbar roots and the first sacral root were divided, the superior border of the anæsthesia corresponds to the inferior border of the second lumbar dermatome (fig. 19), and in a case of resection of the eleventh and twelfth thoracic and the first lumbar roots the inferior border of the anæsthesia represents the superior border of the second lumbar dermatome (fig. 20).

Third lumbar dermatome.—The third lumbar dermatome was outlined by the isolation method on the other leg of the same patient as the second lumbar dermatome (fig. 17). On comparison of the legs the great extent of overlap becomes evident. The third lumbar dermatome has a characteristic shape; a central part around the knee, an upper prolongation along the anterior and internal surface of the thigh, and an inferior strip or band along the inner side of the leg reaching below as far as the internal malleolus. There is also a small band on the dorsal aspect of the trunk, which is not in continuity with the main

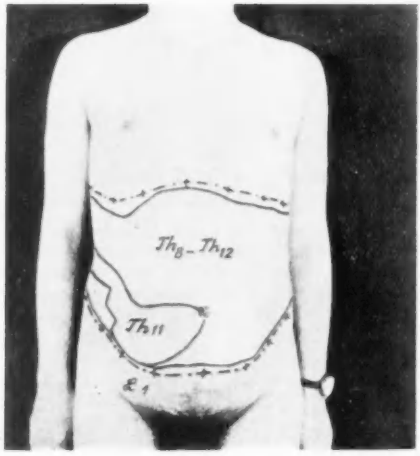


FIG. 12.—The superior border of the 1st lumbar dermatome; resection of 8th to 12th thoracic roots.

area upon the lower limb (fig. 18). Fig. 21 shows the third lumbar dermatome in another case. On comparing these cases it is seen that the extent of the same dermatome may present considerable individual variations. It is noteworthy that in the case represented by fig. 21 the first sacral dermatome, which was also defined, had an unusually large extension. The situation and the extent of the third lumbar dermatome according to Head is shown in fig. 5.

The constructive method confirms the results obtained by the isolation method. In a case in which all the roots between the ninth thoracic and the second lumbar were divided, the inferior border of the anæsthetic area corresponds exactly with the superior border of the dermatome demonstrated above by the isolation method (fig. 22); and

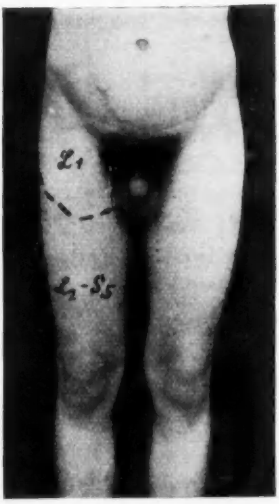


FIG. 13.

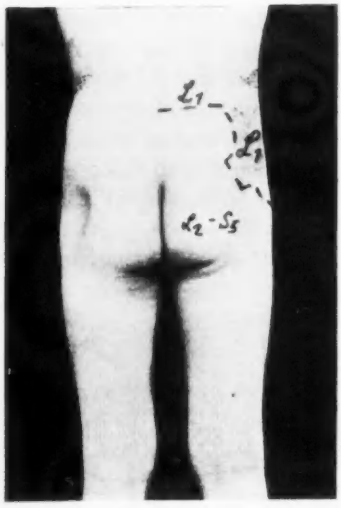


FIG. 14.

FIGS. 13 and 14.—The inferior border of the 1st lumbar dermatome; resection of 2nd to 5th lumbar and 1st to 5th sacral roots.

FIG. 15.

FIG. 16.

FIGS. 15 and 16.—The inferior border of the 1st lumbar dermatome; resection of 2nd and 3rd lumbar roots.

FIG. 17.

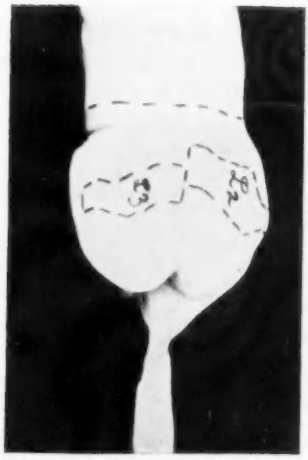


FIG. 18.

FIGS. 17 and 18.—The 2nd lumbar dermatome; resection of Th12 and L1, and L3 to S5 on the right side. The 3rd lumbar dermatome; resection of Th12, L1 and L2, and L4 to S5 on the left side.

FIG. 19.

FIG. 20.

FIG. 19.—The inferior border of the 2nd lumbar dermatome; resection of L3 to S1.
FIG. 20.—The superior border of the 2nd lumbar dermatome; resection of Th11, Th12 and L1.

in another case in which the fourth and fifth lumbar and all sacral roots were divided, the superior border of the anæsthesia runs along the anterior side of the tibia down to the internal malleolus and then upwards along the internal side of the leg (figs. 23 and 24).

The area of vaso-dilatation following electrical stimulation of the

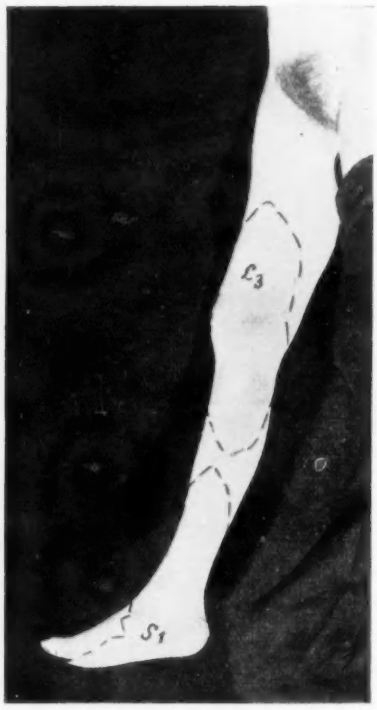


FIG. 21.

FIG. 22.

FIG. 21.—The 3rd lumbar dermatome of relatively small extent. The 1st sacral dermatome of large extent. Resection of Th11 to L2, L4 and L5, S2 to S5.

FIG. 22.—The superior border of the 3rd lumbar dermatome; resection of Th9 to L2.

third lumbar root (fig. 25) also coincides in situation and shape with the dermatome defined by the isolation method, but its extent is not quite as large as that of the area of remaining sensibility.

Fourth lumbar dermatome occupies the anterior side of the leg, the internal part of the dorsum of the foot and the great toe (fig. 26). On the sole its border runs from between the first and second toes back-

wards behind the internal malleolus (fig. 27), and upwards along the posterior-internal side of the leg. At the level of the knee it turns forwards, then bends behind the head of the fibula to the outer side of the leg (fig. 28) and finally returns to the dorsal aspect of the foot where it reaches the interdigital space between the hallux and the second toe.

FIG. 23.

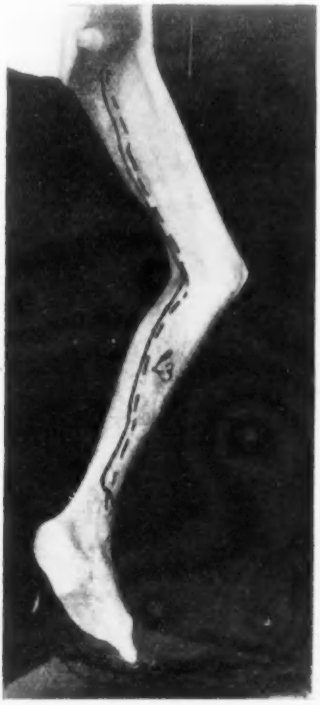


FIG. 24.

FIGS 23 and 24.—The inferior border of the 3rd lumbar dermatome; resection of L4 to S5.

In the second case the situation, shape and extension of the fourth lumbar dermatome were almost identical (figs. 29, 30 and 31). In a third case the extent was very similar (figs. 32 and 25). The photograph (fig. 25) shows the large overlap of the fourth and the third lumbar dermatomes, the latter of which was outlined by the vaso-dilatation excited by faradic stimulation of the third lumbar root. The topography of the fourth lumbar dermatome in a fourth case is shown in fig. 33.

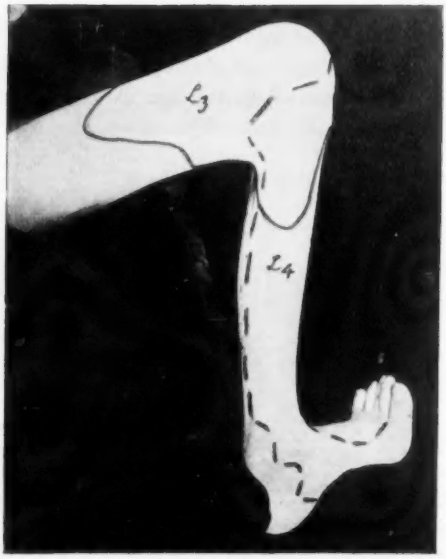


FIG. 25.—The 3rd lumbar dermatome defined by vasodilatation following electrical stimulation of 3rd posterior lumbar root. The 4th lumbar dermatome defined in the same case by remaining sensibility after resection of all lumbar and sacral roots except the 4th lumbar. The continuous line represents the border of the 3rd dermatome, the broken line that of the 4th lumbar dermatome.

FIG. 26.

FIG. 27.

FIG. 28.

FIGS. 26, 27 and 28.—The 4th lumbar dermatome; resection of L2 and L3, and L5 to S5.

The fourth lumbar dermatome, as defined by the isolation method, has a markedly larger extent than Head determined. In Head's scheme the big toe is not included in it.

The fifth lumbar dermatome occupies the anterior side of the leg, the entire dorsum of the foot and of all toes (figs. 34, 35 and 36). The

Fig. 29.

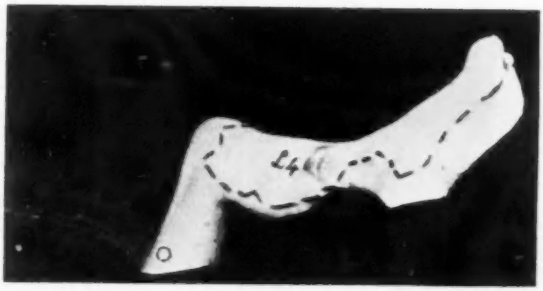


Fig. 30.

Fig. 31.

Figs. 29, 30 and 31.—The 4th lumbar dermatome in a second case; resection of L2 and L3, and L5 to S5.

border runs along the outer border of the foot behind the external malleolus, up the leg and then turns below the head of the fibula to the anterior aspect, from where it runs down along the internal side of the leg to the sole. The dermatome occupies the medial part of the sole, and the plantar surfaces of the first, second and third toes, but not that of the fourth and fifth toes (fig. 37).

The constructive method reveals the same borders for the fifth lumbar dermatome. In a case in which I resected the first, second and

third sacral roots, the anæsthetic area on the posterior side is shown in fig. 38. Its border runs along the external side of the foot, but the medial half of the sole and the first, second and third toes preserved their sensibility (fig. 39). In this photograph the continuous line indicates the limits of tactile anæsthesia, the broken line that of the analgesia; the extent of the latter is larger than that of the former. This is common in cases of root resection, and corresponds with the

Fig. 32.

Fig. 33.

Fig. 32.—The 4th lumbar dermatome in a third case; resection of L2 and L3, and L5 to S5.

Fig. 33.—The 4th lumbar dermatome in a fourth case; resection of L2 and L3, and L5 to S5.

fact, pointed out by Sherrington, that the tactile dermatomes are larger than the pain dermatomes.

The first sacral dermatome occupies the sole and the plantar surfaces of the toes (fig. 40); in some cases it extends even to the dorsal surfaces of the toes. From the sole the dermatome spreads upwards along the posterior side of the leg (fig. 41). The size and shape of the first sacral dermatome, as defined by the isolation method, are very similar to the first sacral dermatome in Head's scheme (fig. 5).

The second sacral dermatome occupies the posterior surface of the

thigh and of the leg, the sole of the foot and the plantar aspects of the toes (fig. 42). In another case the second sacral dermatome extended on to the dorsal surfaces of the toes (fig. 19). The extent of the remaining sensibility in my cases was considerably larger than the area of the second sacral dermatome in Head's scheme (fig. 5).

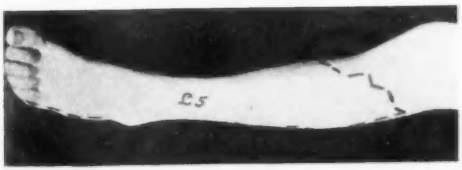


FIG. 34.

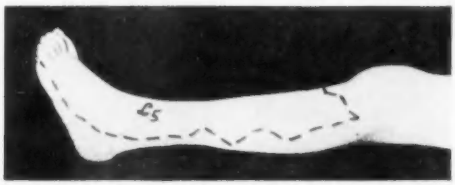


FIG. 35.

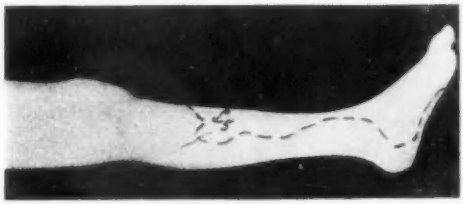


FIG. 36.

FIG. 37.

FIGS. 34, 35, 36 and 37.—The 5th lumbar dermatome : resection of Th12 to L4, and S1 to S5.

THORACIC DERMATOMES.

I have had no opportunity of defining the different thoracic dermatomes by the isolation method, but the constructive method has furnished all the details necessary for determining the inferior and superior borders of practically all of them. A full description of all these separate dermatomes is not necessary ; their distribution and size can be seen in the diagram which I have worked out from the

examination of a great number of cases of division of posterior thoracic roots (fig. 43). A few details are, however, worthy of notice.

The tenth thoracic dermatome has a typical situation around the umbilicus, and the superior border of the eleventh and the inferior border of the ninth thoracic dermatomes extend just to it. The mammilla lies within the fifth thoracic dermatome, and the inferior border of the fourth and the superior border of the sixth reach to a horizontal line through it.

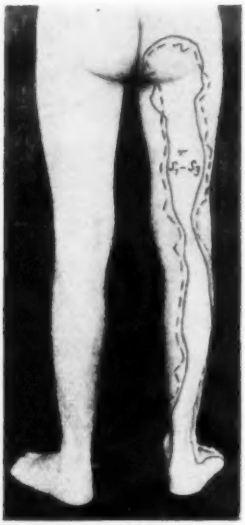


FIG. 38.

FIG. 39.

FIGS. 38 and 39.—The anæsthesia following resection of S1, S2 and S3, showing the border of the 5th lumbar dermatome on the planta pedis; the tactile dermatome (continuous line) is of larger extent than pain dermatome (broken line).

The thoracic dermatomes, as defined by the vasodilatation excited by electrical stimulation of the posterior thoracic roots, conform closely to the areas obtained by the constructive method (figs. 12, 44, 45, 46, 47, 48, 49, 50).

The area of vasodilatation following faradic stimulation of the third thoracic root includes a triangular patch in the axilla. In the case represented by fig. 51 the latter was not continuous with the main area upon the thorax. I have found this lack of continuity of the areas of vasodilatation following electrical stimulation of the posterior roots in a good many other cases.

In another case the triangular area in the axilla was not so large (fig. 52); there exist undoubtedly considerable individual variations in it. A similar triangular prolongation of the third thoracic dermatome into the axilla is demonstrable by the constructive method, as after division of the first and second thoracic roots the area of resulting

FIG. 40.

FIG. 41.

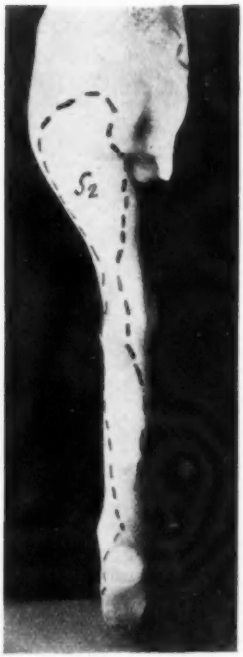


FIG. 42.

FIGS. 40 and 41.—The 1st sacral dermatome and the 3rd lumbar dermatome; resection of Th11 to L2, L4 and L5, and S2 to S5. Compare fig. 21.

FIG. 42.—The 2nd sacral dermatome; resection of Th11 to S1, and S3 to S5.

anæsthesia shows the characteristic indentation due to its prolongation into the axilla (fig. 53). In another case of resection of the first and second thoracic roots the characteristic triangular prolongation of the third thoracic dermatome also is visible (fig. 54), and in another patient in whom the second and first thoracic, and the eighth and seventh cervical roots were resected, the triangular prolongation of the third thoracic dermatome into the axilla comes out very clearly (fig. 55).

Sometimes this triangular prolongation of the third thoracic dermatome extends even further down the arm, as in a case of resection of the second and first thoracic roots and the fifth to the eighth cervical roots reported by Head (fig. 56). In this case the triangular area of remaining sensibility resembles closely in size and shape the triangular prolongation delimited by the area of vaso-dilatation produced by electrical stimulation of the third thoracic root, which is shown in one of the illustrations (fig. 51).

The second thoracic dermatome, as represented by the area of vaso-dilatation excited by faradic stimulation of the second thoracic root,

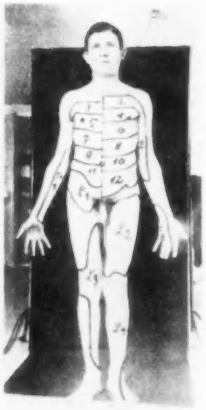


FIG. 43.

FIG. 43.—Scheme of thoracic dermatomes.

occupies the inner side of the arm. It extends downwards as far as the internal condyle of the humerus, and it is prolonged on to the anterior and posterior surfaces of the thorax (figs. 57, 58). In the case illustrated by fig. 58, vaso-dilatation reached as far as the wrist; this was due to the fact that the fibres of the first thoracic dermatome were also stimulated in consequence of an anastomosis between the second and the first thoracic roots. The two bands of the second thoracic dermatome which extend on to the thorax from the inner side of the arm form a characteristic angle (fig. 58c), and consequently in all cases of division of the third, fourth and fifth thoracic roots the anæsthesia includes a triangular extension into the axilla (fig. 59).

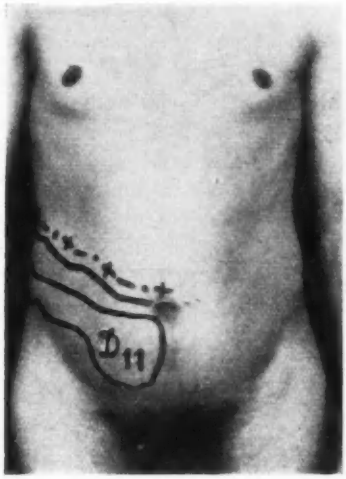


FIG. 44.

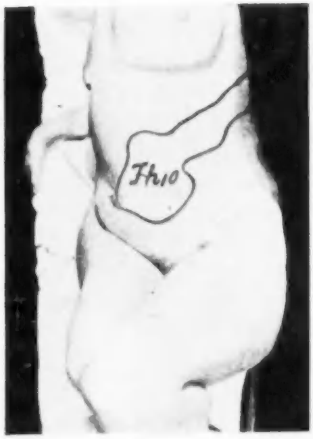


FIG. 45.

FIG. 44.—The 11th thoracic dermatome defined by vasodilatation; resection of Th10 to S5.

FIG. 45.—The 10th thoracic dermatome defined by vasodilatation.

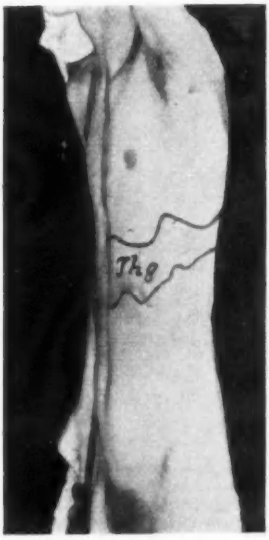


FIG. 46.

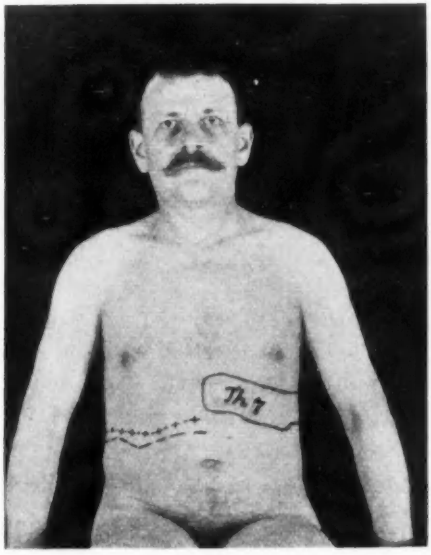


FIG. 47.

FIG. 46.—The 8th thoracic dermatome defined by vasodilatation.

FIG. 47.—The 7th thoracic dermatome defined by vasodilatation.

The first thoracic dermatome, as represented by the area of vasodilatation following electrical stimulation of the first thoracic root, occupies the internal side of the fore-arm as far down as the wrist (fig. 57). Unfortunately the superior border of this dermatome could not be mapped out in this case, since the second thoracic root had been stimulated just before and the effect of this stimulation, which always lasts a considerable time, had not disappeared when the first thoracic root was stimulated.

FIG. 48.

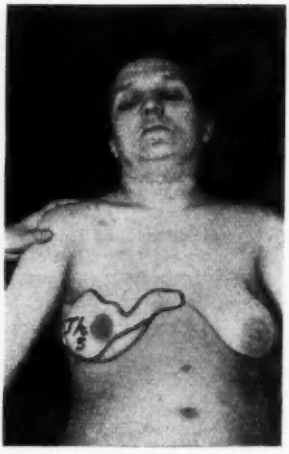


FIG. 49.

FIG. 50.

FIGS. 48 and 49.—The 5th thoracic dermatome defined by vasodilatation.
FIG. 50.—The 4th thoracic dermatome defined by vasodilatation.

That the second thoracic root, and probably the first thoracic root as well, take part in the innervation of the thorax, is demonstrated by the fact that after resection of the first and second thoracic roots a small band of anæsthesia is, in some cases, found on the thorax (fig. 60).

CERVICAL DERMATOMES.

The eighth cervical dermatome, as represented by the area of vasodilatation following electrical stimulation of the eighth cervical root, occupies the fifth and fourth fingers, the ulnar half of the third finger,

FIG. 51.

FIG. 52.

FIG. 51.—The 3rd thoracic dermatome defined by vasodilatation.

FIG. 52.—The 3rd thoracic dermatome defined by vasodilatation.

FIG. 53.

FIG. 54.

FIG. 53.—Anæsthesia following resection of Th1 and Th2 with the characteristic indentation of the 3rd thoracic dermatome in the axilla. Note the considerably larger extent of analgesia and thermanæsthesia + −. + −., than that of the tactile anæsthesia, ——.

FIG. 54.—Anæsthesia following resection of Th1 and Th2; indentation by the 3rd thoracic dermatome in the axilla. Analgesia and tactile anæsthesia of about the same extent.

the ulnar part of the hand and to some extent the distal part of the fore-arm (fig. 61). The overlap of the eighth cervical dermatome on the fore-arm is demonstrated by a case in which the first and second thoracic roots had been resected ; the inferior border of the anæsthesia does not reach as far as the wrist (fig. 62), and the sensibility preserved on the lower part of the fore-arm must be due to extension of the eighth cervical dermatome to the fore-arm. Individual variations in the distribution of the eighth cervical dermatome exist however, for in another case of

FIG. 55.

FIG. 56.

FIG. 55.— Anæsthesia following resection of C7, C8, Th1 and Th2, with characteristic indentation by the 3rd thoracic dermatome in the axilla. Analgesia and thermanæsthesia + −, + −, of larger extent than tactile anæsthesia, ———.

FIG. 56.— Tactile anæsthesia ..... and analgesia ——— following resection of C5 to Th2 reported by Head. The 3rd thoracic dermatome occupies the internal surface of upper arm.

resection of the first and second thoracic roots, already demonstrated above, the anæsthesia reached distalwards as far as the wrist (fig. 53).

I have no cases affording landmarks for the definition of the seventh cervical dermatome.

The sixth cervical dermatome, outlined by the remaining sensibility after resection of the first and second thoracic, and the eighth, seventh, fifth and fourth cervical roots, occupies the thumb, the index finger and the thenar eminence, and extends up the external side of the fore-arm

and of the upper arm (figs. 63, 64). The tactile dermatome is considerably larger than the pain dermatome.

In all cases in which the first, second, third, fourth and fifth cervical roots are divided, the inferior border of the anæsthesia outlines a triangular flap below the shoulder (fig. 65). The borders of this area represent the superior limits of the sixth and seventh cervical dermatomes.

The fifth cervical dermatome, as shown by the vaso-dilatation following stimulation of the fifth cervical root, covers a large area. It reaches from the shoulder along the lateral side of the arm downwards as far as the thumb (figs. 66, 67, 68). The same case demonstrates very well the large overlap of the fifth cervical dermatome with the sixth and

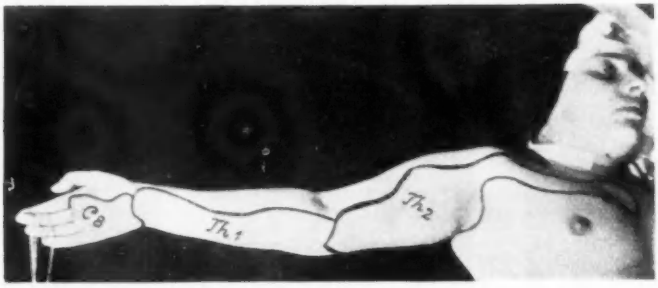


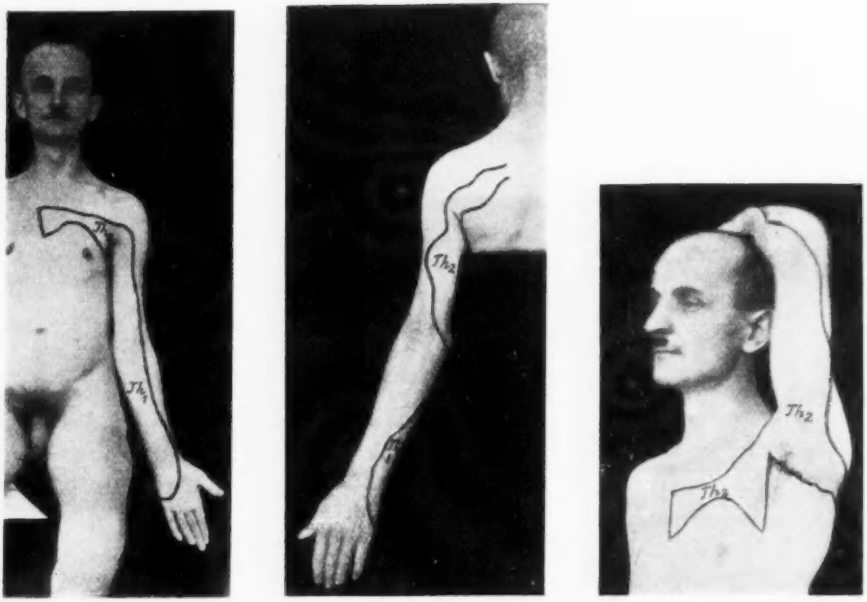
FIG. 57. – The 2nd and 1st thoracic and 8th cervical dermatomes defined by vaso-dilatation. The 1st thoracic and the 8th cervical dermatomes do not show their full extent.

seventh dermatomes; the first, second, third, fourth and fifth cervical roots were resected, but the resulting anæsthesia does not extend further downwards than about 6 cm. below the shoulder; on the other hand the fifth dermatome reaches as far as the thumb.

The fourth cervical dermatome.—The inferior border of the area of vaso-dilatation following electrical stimulation of the fourth cervical root is shown in fig. 69. Its superior border could not be outlined in this case as the skin of the neck had been coloured by a solution of iodine. In this case the first, second, third and fourth cervical roots were resected. Fig. 70 shows that the area of vaso-dilatation produced by faradic stimulation of the fourth cervical root extends much lower than the inferior border of the area of anæsthesia following the resection of the first to fourth cervical roots. This shows clearly the amount of

overlap of the fourth cervical dermatome with the fifth, sixth and seventh cervical dermatomes and the first and second thoracic.

The constructive method confirms the result obtained by the electrical stimulation of the fourth cervical root. In a case (figs. 71 and 72) in which the fifth, sixth, seventh and eighth cervical, and the first, second and third thoracic roots were divided, the characteristic



A B C

FIG. 58.—The 2nd and 1st thoracic dermatomes defined by vasodilatation.

triangular area of the fourth cervical dermatome may be seen at the superior border of the resulting anæsthesia.

In a case reported by Cushing, in which the second and third cervical roots were resected, the inferior border of the anæsthesia marks the superior border of the fourth cervical dermatome (fig. 73). My own cases of resection of the second and third cervical roots demonstrate a similar inferior border of the anæsthesia (figs. 74, 75). The analgesia occupies a larger area than the tactile anæsthesia, that is to say the pain dermatome is considerably smaller than the tactile. The difference between the extent of analgesia and that of tactile anæsthesia is still

greater in another case in which the second and third cervical roots were resected (figs. 76, 77); the area of anæsthesia is exceptionally small, but the inferior border of the analgesia and thermanæsthesia conforms to Cushing's observations.

FIG. 59.—The superior border of the anæsthesia in a case of chordotomy interrupting the pain and thermal fibres corresponding to the 3rd, 4th, 5th and 6th and lower thoracic roots.

FIG. 60.—The anæsthesia following resection of 7th and 8th cervical, and 1st and 2nd thoracic roots. Note the small band of analgesia and thermanæsthesia upon the chest.

The third cervical dermatome, as represented by the area of vasodilatation following electrical stimulation of the third cervical root, is shown in fig. 78. It reaches upwards to the mandible, includes the ear and extends downwards as far as the clavicle. In the same case the

fifth cervical root also was stimulated, and it can be seen how closely the two areas of vasodilatation come together.

The inferior border of the third cervical dermatome was also defined by resection of the fifth and the fourth cervical roots (fig. 79). The case is interesting in as far as there was no tactile anæsthesia ; the resection of the two roots produced analgesia and thermanæsthesia only, further evidence of the larger extent of the tactile than of pain and thermal

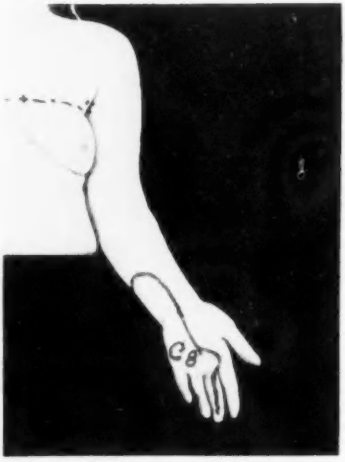


Fig. 61.

Fig. 62.

Fig. 61.—The 8th cervical dermatome defined by vasodilatation.

Fig. 62.—Anæsthesia following resection of Th1 and Th2. Note the conservation of sensibility in the interior part of the fore-arm. Compare fig. 54.

dermatomes. In another case in which the fourth, fifth, sixth, seventh and eighth cervical and the first to the fifth thoracic roots were resected the lower border of the third cervical dermatome was similar (fig. 80), but sometimes the third cervical dermatome extended further downwards, as in a case in which the fourth, fifth, sixth, seventh and eighth cervical and the first to the fourth thoracic roots were resected (fig. 81). This case demonstrates again the considerable individual variations in the extent of a single dermatome.

FIG. 63.

FIG. 65.

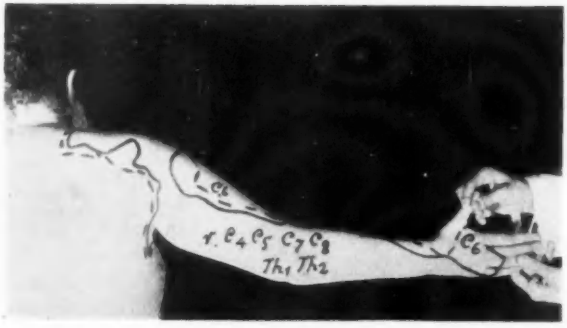


FIG. 64.

FIGS. 63 and 64.—The 6th cervical dermatome; resection of C4 and C5, and of C7 to Th2.

FIG. 65.—The anæsthesia following resection of C1 to C5.

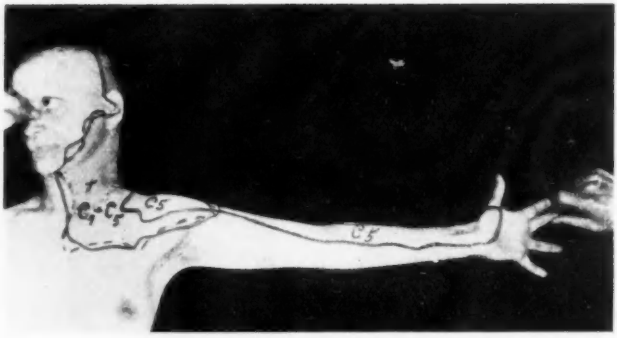


FIG. 66.

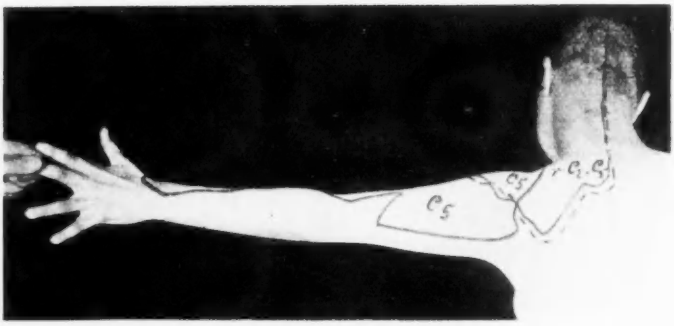


FIG. 67.

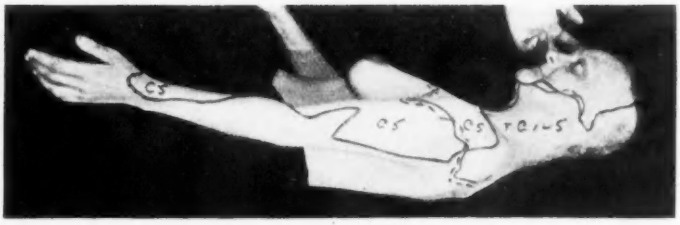


FIG. 68.

FIGS. 66, 67 and 68.—The 5th cervical dermatome defined by vasodilatation and the anæsthesia following resection of C1 to C5.

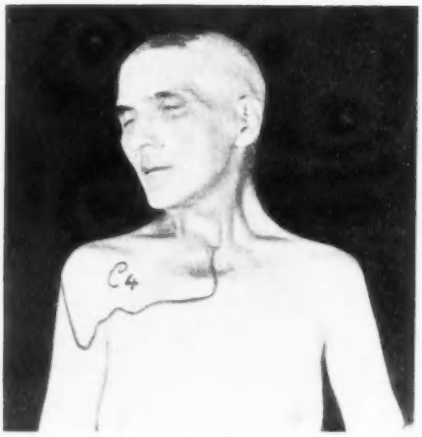


FIG. 69.

FIG. 70.

FIG. 69.—The inferior border of 4th cervical dermatome, defined by vasodilatation.

FIG. 70.—The inferior border of 4th cervical dermatome, defined by vasodilatation, and the inferior border of the anæsthesia following resection of C1 to C4. This figure demonstrates the large overlap of the 4th cervical dermatome on the adjacent dermatomes.

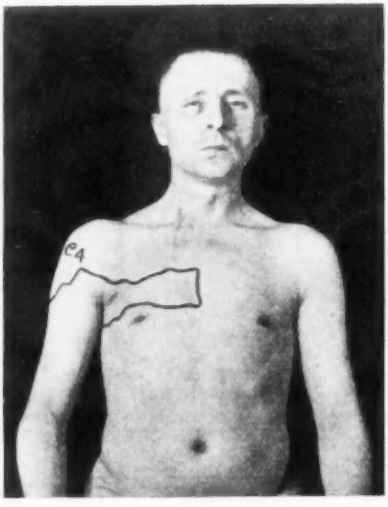


FIG. 71.

FIG. 72.

FIGS. 71 and 72.—The superior border of the anæsthesia, following resection of C5 to Th4. It demonstrates the inferior border of 4th cervical dermatome.

The second cervical dermatome occupies the occiput and the submental region, as may be seen in a case in which all cervical roots except the second were divided (figs. 82, 83). The anæsthesia involves the ear and sends a triangular projection towards the temporal region. But there

FIG. 73.—The inferior border of the anæsthesia following resection of C2 to C3 (according to Cushing). It demonstrates the superior border of the 4th cervical dermatome.

FIG. 74.

FIG. 75.

FIGS. 74 and 75.—The anæsthesia following resection of C2 and C3. The inferior border of the anæsthesia indicates the superior border of 4th cervical dermatome. Note the much larger extent of the analgesia – – – than of the tactile anæsthesia ———.

exist considerable individual variations in the extent of the second cervical dermatome, for in a case in which the same roots were resected and the second cervical root alone was left intact, the ear is partially included in the anæsthesia (fig. 84); on the other hand the loss of

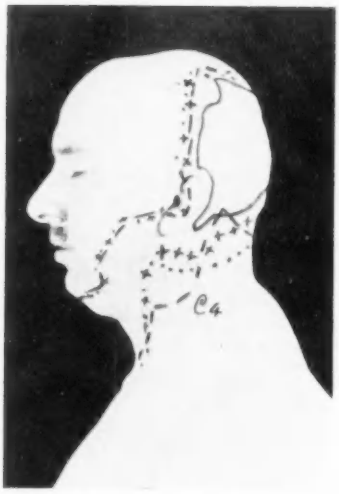


FIG. 76.

FIG. 77.

FIGS. 76 and 77.—The anæsthesia following resection of C2 and C3. The inferior border indicates the superior border of the 4th cervical dermatome. Note the small extent of tactile anæsthesia ———, the much larger extent of the analgesia – – –, and of thermanæsthesia +. +. +.

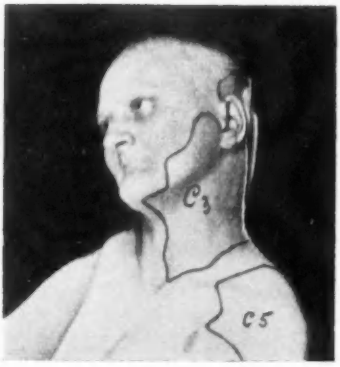


FIG. 78.

FIG. 79.

FIG. 78.—The 3rd cervical dermatome defined by vasodilatation.

FIG. 79.—The inferior border of the 3rd cervical dermatome defined by the superior border of the sensory loss following resection of C4 and C5. Note the complete absence of tactile anæsthesia in this case.

FIG. 80.

FIG. 81.

FIG. 80.—The inferior border of the 3rd cervical dermatome defined by the superior border of the anæsthesia following resection of C4 to Th5.

FIG. 81.—The inferior border of the 3rd cervical dermatome defined by the superior border of the anæsthesia following resection of C4 to Th4. Note the large extent of the 3rd cervical dermatome upon the thorax.

FIG. 82.

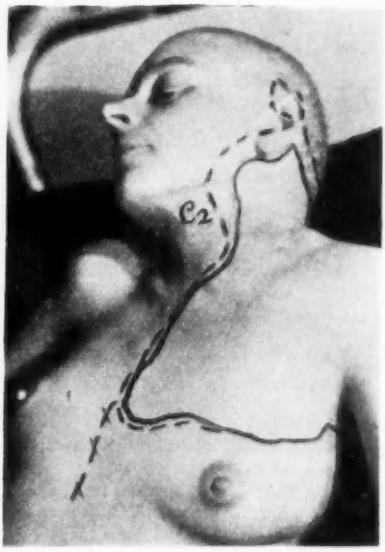


FIG. 83.

FIGS. 82 and 83.—The 2nd cervical dermatome defined by the superior border of the anæsthesia following resection of all the cervical roots except the second.

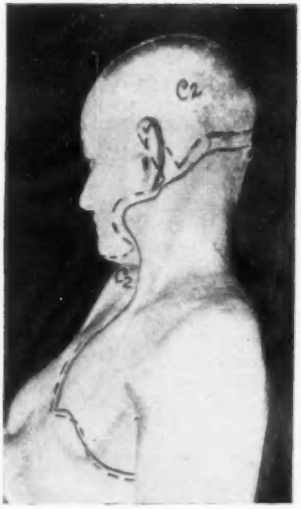


FIG. 84.

FIG. 85.

FIGS. 84 and 85.—The 2nd cervical dermatome defined by the superior border of the anæsthesia following resection of all the cervical roots except the second.

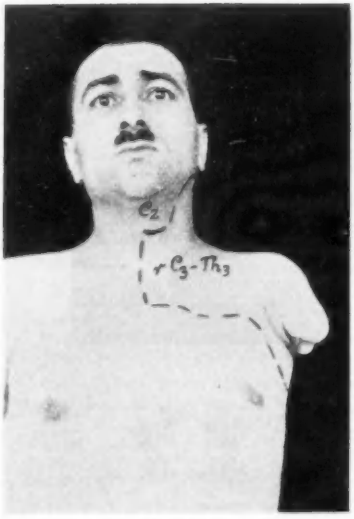


FIG. 86.

FIG. 87.

FIGS. 86 and 87.—The 2nd cervical dermatome defined by the superior border of the anæsthesia following resection of all the cervical roots except the second.

sensation, at least as far as pain sensibility was concerned, extended into the submental region (fig. 85). In a third case of resection of all cervical roots except the second, the second cervical dermatome occupied

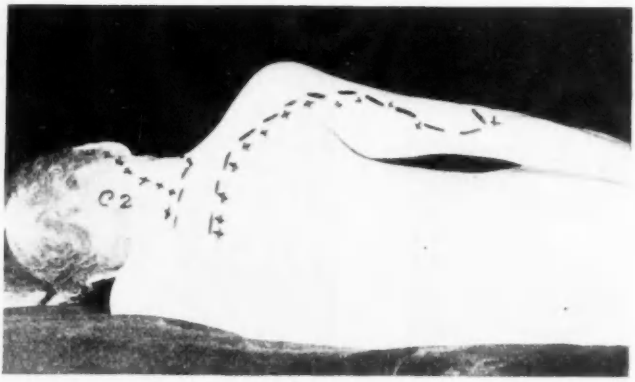


FIG. 88.

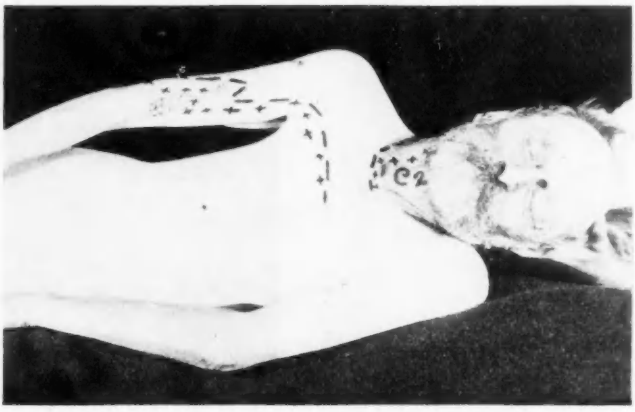


FIG. 89.

FIGS. 88 and 89.—The 2nd cervical dermatome defined by the superior border of the anæsthesia following resection of C3, C4, C5 and C6.

the occiput, a large area on the neck and the submental region (figs. 86, 87). The distribution of the anæsthesia was similar in a further case in which the third, fourth, fifth and sixth cervical roots were divided; here the second cervical dermatome extends from the occiput

far down on the neck and occupies a large area on its anterior surface (figs. 88, 89).

The oral border of the second cervical dermatome is represented by the border of anæsthesia following resection of the fifth cranial nerve (fig. 90). In the majority of my cases the line separating these two areas runs down from the vertex anteriorly to the ear, but the tragus is not included in the anæsthesia, and I have seldom found the helix

Fig. 90.

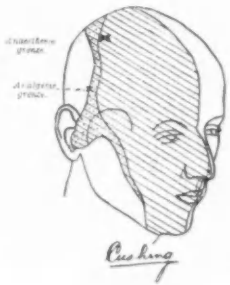


Fig. 91.

Fig. 90.—The superior border of the 2nd cervical dermatome defined by the caudal border of the anæsthesia following resection of the 5th cranial nerve.

Fig. 91.—The tactile anæsthesia and analgesia following division of the trigeminal root, according to Cushing.

anæsthetic. But the observations of other investigators indicate that individual variations exist. Fig. 91 shows the area of anæsthesia following section of the trigeminal root according to Cushing; the tragus is included within it.

The caudal border of the dermatomes corresponding to the cranial nerves, if such an expression be allowed, is represented, according to my experience, by a very typical line. I have been able to determine this in twelve cases in which I resected all the upper cervical roots (figs. 92, 93). In all these cases the anæsthesia included the ear, but the external

meatus and the skin around it were not anæsthetic. The individual variations are slight. I believe that it is not correct to say that this border is the caudal limit of the skin supply of the fifth cranial nerve, for we must bear in mind that the seventh, ninth and tenth cranial nerves participate to some degree in the supply of the ear.

FIG. 92.

FIG. 93.

FIGS. 92 and 93.—The superior border of the anæsthesia following resection of all the cervical roots; it indicates the caudal border of the cranial nerve supply to the skin.

CONCLUSIONS.

From the material at my disposal several conclusions seem to me permissible.

(1) The dermatomes of man overlap to the same large degree as do those of the monkey. I never have found that resection of one single root in man was followed by loss of sensibility; at least by the usual clinical methods of examining sensibility no defect could be detected. When a single posterior root has been divided it is only by counting carefully the number of pressure and pain spots in the area innervated by that root, and by measuring exactly the threshold of the spots for mechanical or electrical stimuli, that a diminution in the number of root fibres connected with the skin can be demonstrated.

(2) The tactile dermatomes are larger than the pain and the thermal dermatomes. The area of analgesia and thermanæsthesia following resection of a number of contiguous roots is always larger than the area of tactile anæsthesia. Sometimes the resection of two contiguous roots produces analgesia and thermæsthesia, only, but no tactile anæsthesia.

(3) The areas of vasodilatation produced by electrical stimulation of posterior roots are similar to the dermatomes defined by the isolation method, but not identical with them. The former are very similar to the areas of herpetic eruption.

(4) One last word to illustrate the conformity between the dermatomes of man and monkey. A single dermatome is represented not only in the totality of the corresponding root, but in every single filament of the root. When all filaments except one of each of a number of roots are cut, no anæsthesia results, each of the corresponding dermatomes preserves its sensibility. This was demonstrated in animals by several investigators. The same is to be observed in man. I have resected in several cases about two-thirds and even more of each of all the lumbar and sacral roots, leaving intact a small filament only of each root. In these cases the sensibility was preserved.

(5) The difference between the results of experimental physiology in animals and clinical observation in man lies in the far greater difficulties presented by human material. The physiologist can do what he believes to be necessary for his investigation; the activities of the neurologist are limited to procedures which are necessary to help his patient, and he can do no more. This difference explains why the physiology of the nervous system of man lags far behind the experimental physiology of animals, and needs more time to achieve a result, which even when obtained will be a modest one.

The material which I have brought before you is the result of thirty years of neuro-surgery, and at the end I feel that I am at about the same milestone as at the beginning. I realize that all our present knowledge of the dermatomes in man has added little to the foundation which was laid just forty years ago by Henry Head in his doctorial thesis in Cambridge, June 1892.

It has given me much pleasure to be invited to deliver this lecture in memory of Dr. Schorstein, who was an eminent physician on the Staff of the London Hospital, more especially as it gives me the opportunity of paying a tribute to his colleague, Sir Henry Head, whose work on the dermatomes and other subjects has added distinction to English neurology.

OBSERVATIONS UPON THE FORM AND NATURE OF THE "GRASPING" MOVEMENTS AND "TONIC INNERVATION" SEEN IN CERTAIN CASES OF LESION OF THE FRONTAL LOBE.

BY F. M. R. WALSHE AND E. GRAEME ROBERTSON.

(From the National Hospital, Queen Square, London.)

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INTRODUCTION.

THE group of motor disorders that have been described under the various titles of "tonic innervation," "the grasp reflex," "forced grasping and groping," and under comparable descriptive names by writers in other languages, has been the subject of growing interest within recent years.

In each succeeding description they have assumed more complex features, until by some writers they have come to be endowed with qualities which conform to no known natural laws. But while the different studies have been as fruitful of nomenclature as of hypotheses, they have not achieved general agreement upon essential clinical details, nor have they removed the great obscurity which still surrounds the origin and nature of the disorders in question.

Therefore, a fresh clinical study needs no apology.

While it is not proposed to preface it with yet another review of the relevant literature, a brief summary of the recorded facts of observation will serve to indicate the present state of opinion.

In 1914, Wilson and Walshe [17] described a disorder of movement which they called "tonic innervation." It was observed in the paretic arms of patients with frontal lobe lesion associated with hemiparesis. They found that voluntary flexion and extension of the arm and voluntary grasping were followed by considerable delay in relaxation, and in the case of the hand by an inability to release the grasp of any object held in it. Any attempt by patient or by observer to extract the object led at once to greatly increased force of grasp, a tonic contraction of the muscles concerned which persisted until the attempt to extract the object ceased. When the grasp was not forced in this way, the hand frequently opened without difficulty. No reflex factor in the initiation of this tonic innervation was detected.

In 1923, Riddoch and Brain [10], in describing a comparable state of affairs, suggested that a palmar reflex might be in question, the stimulus being a touch on the palm.

Almost at the same time, Schuster [12], and later Schuster and Pinéas [13, 14], published lengthy studies of these grasping and tonic phenomena, which they also noted in the paretic arms of patients with frontal lobe lesions. They found as follows:—

(1) That the hand closed over and grasped any object laid upon the open palm.

(2) That the hand grasped involuntarily and even against the patient's will at any object, such as the examiner's finger, that touched the palmar aspect of the hand or digits, and that the hand and arm turned actively towards the stimulating object, following this as though it were a magnet.

(3) That the hand involuntarily grasped at any object brought near it, even before contact with the palm was made.

(4) That the hand involuntarily grasped at and followed an object that had been withdrawn from its grasp or from its neighbourhood.

(5) That any attempt to extricate an object from the grasping hand led to a strong tonic grasp.

(6) That when the clenched hand was empty—whether actively or passively closed—voluntary relaxation was easy and no tonic grasp ensued.

Schuster concluded that the grasping movements ("Zwangsgreifen und Nachgreifen") were involuntary and not within the patient's control, and that the exciting stimulus was the contact of some object with the palm or fingers. He explained the tonic grasp as a kind of tetanic fusion

of individual grasping movements elicited by the rhythmically repeated tactile stimuli delivered by the object contained within the hand.

In 1927, Adie and Critchley [1], in a small series of clinically and pathologically similar cases, confirmed all these findings and amplified them as follows: (1) The grasping movement of the fingers ensues upon any moving contact with the palm, the stimulus being most effective when applied over the metacarpo-phalangeal joints. A touch on thumb or index in this situation evokes a "crab's claw reflex" in which these digits close upon the stimulating object.

(2) The hand, arm and even the trunk are actively turned towards the stimulating object when the palm is touched, and when this is done repeatedly the limb may be drawn in any direction as though by a magnet, and this even when the patient's eyes are closed.

(3) All these complicated movements occur without the knowledge of the patient, who remains unconscious of them, although—paradoxically enough—he is said to be annoyed by them.

(4) Tonic persistence of the grasp ensues when an attempt is made to pull away any object contained in the hand.

(5) Voluntary clenching of the empty hand evokes no tonic grasp and relaxation in these circumstances is rapid and easy.

(6) The patients in whom these movements are seen are psychically normal at the time of their occurrence.

Adie and Critchley believe with Schuster that they are dealing with a reflex response to sensory (tactile) stimulation of the hand, but in respect of the grasping that occurs before the hand has been touched they express no more precise conclusion than that this provides evidence of phenomena "on a still higher plane."

Such, then, is the present state of knowledge, but the attempt to construct from it a complete, consistent and credible picture of the events described is difficult, if not impossible, because so much is left unaccounted for, and because the role attributed to sensory stimulation of the hand is clearly not in harmony with the recorded facts.

For example, we see (1) that tactile stimulation of the hand is not necessary for the occurrence of grasping movements, for the hand grasps an object that is merely seen, but not felt. It moves towards the approaching and follows the retreating hand of the examiner. Such movements through space can occur only in response to stimulation of a distance receptor, such as the eye.

Nevertheless, Adie and Critchley, though not Schuster, state that

these excursions of the limb do occur when the patient's eyes are shut and he is deprived of vision, and that if the hand be repeatedly touched in these circumstances it follows the stimulating object as though this were a magnet. Now a tactile stimulus is effective only at the moment of contact. Before the examiner's finger-tip is actually felt, and after it has left the patient's palm, no stimulus is acting. No one can anticipate the direction from which the stimulating finger is approaching, nor determine the direction in which it is retreating, unless it is seen throughout the whole procedure.

That the hand of the patient without vision should advance to and follow the stimulating finger is not possible. Equally unacceptable is the statement that these very complex movements, even when directed by vision, may be made by a psychically normal individual without his being aware of the fact. More in accord with reason is the statement of Schuster that his patients make these grasping movements in response to an urge to do so, but whether this be the case or not, it seems certain that they must know what is happening.

We see also (2) that tactile stimulation of the hand is effective only under certain special conditions, namely, that the stimulating object must be a foreign body presented in a certain way. For when the patient closes his empty hand, no "forced grasping" ensues and he can open his hand easily. Yet his own finger-tips, when he encloses them in his fist, deliver a sensory stimulus to the palm differing in no single physiological respect from that delivered by the examiner's finger-tips. This observation at once takes the grasping movements out of the category of true reflex movements.

(3) Schuster regards the tonic grasp as the fusion of a series of separate grasping movements ("eine ununterbrochene Aufeinanderfolge einzelner Greifbewegungen") evoked by repeated tactile stimulation from the object contained in the hand. We cannot regard this conclusion as resting upon a sound physiological basis. An object at rest in the hand rapidly ceases to be a potent source of tactile stimuli, and it is difficult to regard the tonic grasp as a kind of tetanus of phasic contractions. It has the characters of a true tonic reflex, a mode of reaction which exteroceptive, tactile stimuli are not adequate to elicit. The possibility that a proprioceptive stimulus may be in question has not been raised by any of the students of this phenomenon, though it leaps to the eye.

(4) Finally, some importance attaches to the psychical condition of the patients in whom these movements occur. Adie and Critchley make

the general statement that this is normal, but reference to the case details given in their paper shows that two of their three patients were grossly abnormal in this respect, while of the third nothing is said as to his condition in this respect at the relevant period of his history. All Schuster's patients were mentally reduced, and when we recall that all cases of the kind have lesions of the frontal lobe—either tumours or softenings within the territory of the anterior cerebral artery—it is not surprising that in fact all show some disorder of volitional control and activity.

Therefore, a survey of the clinical information available indicates that the factors responsible for "forced grasping and groping" still remain to be elucidated, and that some of the recorded facts of observation are themselves suspect.

Chapter I.—Clinical Observations.

If there be any reflex factors in the phenomena we are considering, their presence and characters can be revealed only by a methodical search for the essential components of a true reflex reaction; namely, a specific mode of stimulus, a definite and constant receptive field, and a uniform, co-ordinated motor response. These are the elements of the reflex reaction of the segmental nervous system, with which the researches of Sherrington have made us familiar.

Further, in the event of such a factor being found, we have still to determine whether it includes the whole range of observed movement, or whether it is but a fraction of a larger movement-complex not all of which is reflex in nature.

A preliminary survey of a case presenting the phenomena that we are considering certainly gives the impression that the grasping *movements* with their striking range and delicacy of adaptation from moment to moment, and their apparent independence of sensory stimuli from any single receptive field, are scarcely likely to be true reflexes. On the other hand, the *tonic innervation* of muscles shows a uniformity of behaviour and a dependence upon a single mode of sensory stimulation which suggest strongly that here we are indeed dealing with a reflex reaction of the fixed, inborn variety.

Our own study of a series of eight cases has in fact led us to the conclusion that phenomena of two categories are to be observed, of widely different physiological rank, the one volitional and the other a true reflex reaction.

The clinical material upon which this conclusion is based may now

be described in detail. Not every observed case is reported in full, but such as illustrate the initial evolution of grasping movements and of tonic innervation, their full development and their progressive extinction or dissolution. The cases not reported here are but additional examples and differ in no essential respects from those described.

I.—Cases illustrating the onset and evolution of grasping and of tonic innervation.

Case 1.—H. T., a man, aged 59. (National Hospital, 1933, Dr. F. M. R. Walshe.)

History.—For six months he has been subject to "attacks" in which a peculiar feeling came over him, he flushed and looked vacant, no loss of consciousness. On coming to he said that a sharp pain had darted down the right side of his body into his leg, that the right limbs had stiffened, but had not moved. For the same period he complained of headaches.

For past three months he had noticed that at times he could not "let go" with the right hand when he was holding something in it.

For some months, his wife said, he had been irritable, forgetful and altered in disposition.

A week ago after an "attack" the right leg began to drag.

First examination (January 7, 1933). Fully conscious and co-operative. An unreliable witness, rather fatuous in manner. Speech normal.

Optic discs: slight haziness of upper edges. A small hæmorrhage in left fundus. Quadrantic defect of right visual fields, right and left eyes.

Pupils and ocular movements normal. No facial asymmetry. Tongue protruded straight. Corneal reflexes normal.

Motor system: A slight right hemiparesis, leg most affected, face unaffected. Left limbs normal.

Sensory system: Slight impairment of tactile acuity, of localization, of two point discrimination and of appreciation of size and form in right hand. Painful and thermal sensibility normal. Comparable sensory impairment in right foot.

Reflexes: Tendon-jerks brisker right than left. Transient clonus right ankle.

Abdominal reflexes diminished on right. Right plantar extensor, left flexor.

Right limbs.—Passive movement of the right arm evokes a tonic resistance in biceps and the elbow flexors, a resistance persisting throughout the whole range of movement and until the arm is released. The limb is slightly tremulous in use and finger movements are a little clumsy. No trace of delayed relaxation in voluntary movements.

Grasping movements.—When the observer's fingers are slipped into the patient's right palm between his thumb and index, the patient's fingers close gently on them and hold in a gentle clasp. At request the patient can open his hand freely provided that the fingers in his clasp remain quite still.

When the examiner's index finger is gently introduced between the patient's thumb and index so as to stroke the fold of skin between them, it is met by a gently closing movement of these digits. Unless the skin is touched there is no movement, and there is no movement of the patient's arm through space to meet or follow the examiner's finger.

The patient can prevent the occurrence of the grasping movements described, when asked to do so.

When the examiner's finger is introduced into the patient's palm from the ulnar side, there is usually no grasping, but vigorous rubbing of the palm sometimes evokes a grasping movement of the fingers.

When the patient's hand is turned palm upwards and supported on the palm of one of the examiner's hands, it can be touched with cotton-wool, stroked, pressed and pricked with a pin without any trace of grasping response.

Tonic innervation.—None of the procedures described above evoke any trace of tonic grasp. But this is constantly produced when the examiner pulls his fingers out of the patient's clasp, especially if this is done so as also to pull on the arm. This tonic grasp persists indefinitely and until the pull is released, when he can open his hand easily.

The same tonic response is elicited by placing the handle of a wooden stethoscope transversely within the crook formed by the patient's semi-flexed fingers and giving a sharp pull so as to stretch the flexors of the fingers. The patient cannot prevent the development of this tonic contraction of his finger flexors, and when asked to let go, makes a series of efforts. At first the fingers open out at the metacarpo-phalangeal joints, the interphalangeal joints remaining flexed. Further efforts finally allow these to open after some seconds delay.

He can open his empty fist without any delay or difficulty, and he can also open his grasp with equal ease when it contains the wooden handle of the stethoscope, provided that the latter lies free in his hand.

Occasionally, as the handle drops downwards its bell-shaped end strikes against the side of the patient's thumb and this may elicit a short closing movement of thumb and index. But if a plain straight object be used this does not occur.

Subsequent examinations: During the week after admission to hospital, the headaches and the "attacks" ceased, and all the phenomena described above were unobtainable. Sometimes manipulation of the hand led to the appearance of tonic contraction of the flexors of the fingers to stretch, but no grasping *movements* were obtainable.

He was then given $\frac{1}{100}$ gr. of hyoscine hydrobromide hypodermically. An hour later examination gave the following results. He was a little somnolent and fatuous when aroused, but could be made to co-operate easily.

The right hemiparesis was a little more marked than previously. To stroking and touching of the palm and of the skin between thumb and index there were quick grasping movements, but no tonic innervation. Uncertain grasping to stroking of ulnar side of palm. Sensory testing of the upturned palm as described above gave no trace of grasping movements.

He could open the empty hand, or the hand containing a wooden rod easily.

The flexors of the elbow showed marked tonic resistance to stretching. Tonic innervation to stretching of the finger flexors was well marked and forceful and well sustained.

The patient could prevent the occurrence of grasping movements, but had no vestige of control over the development of tonic innervation of the finger flexors to stretching.

Summary.—A case of right hemiparesis, the leg being most affected, with sensory changes of cortical type and an incomplete right homonymous hemianopia. The motor symptoms suggest the presence of a frontal lobe lesion near the mid-line and vertex. The sensory and visual signs suggest the presence of other lesions. The ophthalmoscopic appearances are compatible with an early papillœdema. At the time of writing it seems probable that multiple tumours are present. Grasping movements and tonic innervation are developing and can be released when absent by the administration of hyoscine. Tonic innervation seems to be the first of these phenomena to appear. It is elicited only by stretch of the muscles concerned. It is not within the patient's control. Grasping movements are obtained to certain specialized tactile stimuli applied to part of the hand, but not to other tactile stimuli of identical physiological quality. These movements can be prevented by the patient.

Case 2.—W. C., a man, aged 55. (University College Hospital, 1933, Dr. F. M. R. Walshe.)

History.—For six weeks he has been subject to frequent attacks, consisting of stiffness of the right limbs, turning of the head to the right and then clonic movements of the right arm and leg. The attack begins in the leg, which is more severely affected than the arm or face. No loss of consciousness, nor of speech during attack. Headaches since first attack, and progressive weakness of right limbs, especially of the leg.

Examination: January 13, 1933.—Fully conscious and co-operative, but euphoric and a bad witness. Memory bad. Occasionally at a loss for a word.

Optic discs normal. Visual fields normal.

Slight right hemiparesis, the leg being most affected, the face least. No sensory loss. Tendon-jerks brisker right than left. Right abdominal reflexes diminished. Right plantar doubtful, left flexor.

Right arm.—Very slight weakness, but no appreciable hypertonus. Finger movements a little clumsy. Passive movement of the arm is unresisted, but quick passive extension of the hand and fingers evokes a distinct tonic contraction in the flexors.

Grasping. When the examiner's index and middle fingers are slipped into

the right palm between thumb and index, the patient's thumb and fingers make a quick grasping movement and hold the examiner's fingers in a gentle clasp. When this procedure is repeated, but the examiner slips his fingers into the palm from the ulnar side of the patient's hand, there is no grasping movement.

If the examiner's index finger repeatedly strokes the fold of skin between the thumb and index of the patient's right hand ; each contact evokes a grasping movement of the patient's thumb and index.

When the patient is told to keep his fingers still under these conditions he can do so without difficulty, but when his attention is distracted the grasping movements resume.

Unless contact is made with the patient's hand there is no grasping, and there is no movement of the hand and arm through space to meet the examiner's hand.

He can open his empty fist quickly and without effort, and if a fountain-pen be placed in his grasp he can open his hand and drop it without delay or difficulty.

When his hand is turned palm up and supported on the examiner's, sensory testing—cotton-wool, stroking and pressure—evokes no trace of grasping.

Tonic innervation.—None of the procedures named above evokes a tonic grasp, but if the attempt is made to pull the fingers or any other object contained in the patient's grasp away—especially if the pull is away from the patient—a tonic contraction of the finger flexors at once ensues and persists until the pull is released, when he can open his hand without difficulty.

If the examiner's index finger or a wooden rod is placed in the curve of the semi-flexed fingers of the patient's hand, and then pulled brusquely so as to stretch the flexors, tonic contraction of these muscles ensues and the patient can be pulled into a sitting position in the bed. He cannot open his fingers until the pull is released.

There was no trace of tonic innervation in the leg.

Summary.—A case presenting the clinical signs of a left frontal neoplasm. Grasping movements of the fingers are elicited by certain forms of tactile stimulation of the hand, but not by others. No tonic innervation is elicited in this way. The patient can readily prevent the occurrence of grasping, and he can readily open the empty hand, or the hand containing an object, provided that the latter is in no way interfered with so as to stretch the finger flexors.

Tonic innervation is elicited by stretch of the finger flexors, and by no other mode of stimulus. It is not within the patient's control.

II.—Cases illustrating fully developed grasping and tonic innervation.

Case 3.—Davis, A., a tailor's cutter, aged 43. (National Hospital, 1931, Dr. Hinds Howell.)

History.—Eighteen months, headache, depression and irritability. Four weeks, apathetic and unable to speak. One week, progressive stupor with incontinence of urine and fæces. Lay motionless in bed staring vacantly before him.

Condition on examination.—Conscious and well oriented. No spontaneity of speech or action, lies motionless and speechless in bed. Occasionally rubs his nose with his left hand. Can answer simple questions, but cannot give any account of himslf. Attention can be aroused, but wanders easily. Passes urine and fæces in bed. No aphasia or apraxia.

Optic discs, bilateral papilloedema. Visual fields normal to rough test. Severe left hemiplegia, with a very slight right hemiparesis. All tendon-jerks brisk, abdominal reflexes lost on both sides. Plantar response extensor on both sides. No evidence of sensory loss.

The left limbs retain a trace of feeble voluntary movement.

Right arm and hand.—(1) The right arm lies on the bed-cover, and when the examiner's right hand approaches it within a few inches, the patient's hand slowly moves to it and encloses it in a gentle clasp. When the patient's eyes are covered this movement does not occur. When his hand meets the examiner's he grasps it entire in slow but normal fashion, moving his hand about until he has secured a comfortable grasp. If one of the examiner's fingers eludes his grasp, he opens his hand normally and fumbles about until he has captured and included it in his grasp. He maintains this grasp in an irregularly waxing and waning fashion, but does not tighten firmly on it. In these circumstances, he can release it readily on demand.

(2) If the examiner's hand is now pulled away, especially if in such a way as to extend the patient's arm and digits, the patient's grasp at once tightens and becomes strong. It is maintained as long as the pull is kept up, and the patient cannot relax voluntarily or to order.

(3) The patient's hand is cupped and supported on the examiner's hand, so that no passive extension of his fingers is possible. Cotton-wool touch, local pressure, pin-prick on the patient's palm evoked no trace of grasping movement. Vigorous rubbing of the palm is also ineffective.

(4) A tonic grasp is immediately provoked by placing the examiner's index finger, or the handle of a wooden stethoscope, at the base of the half-flexed fingers of the patient's hand, and pushing it into the crook formed by the fingers so as to stretch (extend) them. As long as a pull is kept up the tonic grasp is maintained and cannot be voluntarily relaxed.

A tube of very hot water employed in this way evokes a grasp, which is quickly relaxed, the patient rubbing his burning fingers together.

Similar findings were obtained at a subsequent examination.

Post-mortem.—An astrocytoma involving the genu of the corpus callosum and invading both frontal lobes.

Summary.—A case of bilateral frontal lobe lesion.

The hemiparetic right hand and arm deliberately reached to the examiner's hand when this approached his, within his sight, and as deliberately secured a comfortable grasp of it. When the procedure was so arranged as not to awaken the notion of grasping in the patient's mind, various forms of tactile stimulation of the hand failed to evoke grasping. On the other hand, stretching of the finger flexors invariably elicited tonic grasping which was maintained as long as the stretch was kept up.

III.—Cases illustrating progressive dissolution of grasping phenomena and tonic innervation. Catatonia.

Case 4.—H. B., a man aged 69 (National Hospital, 1932, Dr. W. J. Adie.)

History.—Headaches for eighteen months, progressive loss of energy, and apathy for fifteen months, progressive right hemiparesis, confusion, disorientation, drowsiness and double incontinence for seven weeks.

First examination.—Stuporose, easily roused, but readily relapsing into stupor. Optic discs show myopic changes, but no papilloedema. Appears to have useful vision and can fix for a minute or so when aroused and spoken to. Will occasionally answer simple questions slowly. No aphasia, no apraxia of left hand. Small but active pupils.

Severe right hemiplegia, with moderate spasticity, but no voluntary power. The right limbs are in characteristic hemiplegic posture. Tendon-jerks increased on both sides, abdominal reflexes all absent. Right plantar response extensor. On the left, the leg flexes, the hallux plantar flexes and the small toes fan.

Left limbs show no very definite paresis. He occasionally puts his left hand on his nose or head. At times the resting hand gently clasps the bedclothes, but leaves these during movement without any difficulty. To palpation the muscles of the limb are flaccid.

(1) *Passive movement of the arm.*—If the limb is gripped round the wrist and passively extended, biceps and supinator at once go into tonic contraction and stand up under the skin, revealing their contours. There is marked resistance to stretching throughout the range of extension. If the arm is then flexed, a similar, but less marked, resistance is felt in the extensors.

(2) *Grasping movements.*—The patient lies on his back, gazing upwards with eyes half closed. His hand lies on the bed, and is not within his view. The examiner's hand is then moved slowly towards it. It makes no move until touched on the palm. It then slowly clasps the examiner's hand, secures

a comfortable grasp and retains its hold in a waxing and waning grasp, never forceful.

(3) *Tactile stimulation of the palm.*—The patient's left hand is cupped and supported as described above. Cotton-wool touch, pin-prick and local pressure on palm and fingers evoke no grasping movement.

(4) *Tonic innervation.*—The examiner's hand, as it lies within the patient's, is now pulled so as to extend the arm and digits of the patient's limb. A tonic forceful grasp at once develops, and is sustained. The patient's head and back can be pulled off the bed without the grasp relaxing. Brusque orders to him to relax have no effect.

(5) *Left lower limb.*—This lies half flexed and abducted. At times it is slowly extended and again flexed. Passive extension at the knee evokes a strong tonic resistance and the stretched muscles stand out under the skin, as described in the case of the left arm. Stimulation of the sole (stroking movements with finger) evoke a slow plantar flexion and bunching of the

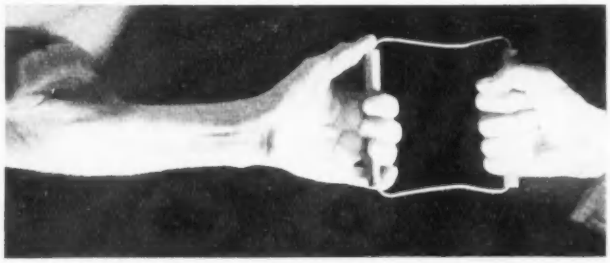

FIG. 1.

digits, and a slow resumption of their original position after two or three seconds. At times the small toes fan instead of plantar flexing, and the limb flexes.

Second examination (November 29, 1932).—Apparently asleep, and with Cheyne-Stokes' respiration. Rouses somewhat during examination, when respiration may become continuous.

During apnœa. The left arm lies flaccid and shows no trace of tonic resistance to passive movement. The left leg is similarly flaccid. On the other hand, the spastic right limbs retain their habitual degree of hypertonus.

During respiration the tonic resistance of the left limbs on passive movement rapidly returns. Also the grasping movements of the left hand described above, which were completely in abeyance during apnœa, return.

If when the examiner's hand is clasped by the patient's, a finger of the examiner's hand eludes his grasp, the patient's hand opens again and his fingers fumble until they have enclosed the complete hand in a comfortable grasp. The tonic grasp can be elicited as on the previous examination.

Third examination (November 30).—Stuporose and in Cheyne-Stokes' respiration.

During apnœa left limbs flaccid and no grasping phenomena or tonic innervation obtainable. Pupils dilate slightly during this phase, and there is also a uniform slight diminution of the tendon-jerks, and plantar stimulation of the left sole evokes dorsiflexion of the hallux.

During respiration the left limbs resume their tonic contraction to stretching, and grasping phenomena-movement and tonic grasping reappear. The hand grasps the examiner's hand, but fails to grasp when given tactile stimuli as described under paragraph 4 in the first examination. Also when clasping the examiner's hand, the patient's hand relaxes quickly if the base of the palm or thumb be pricked with a pin. A tonic grasp can be elicited by the procedure described in paragraph 3 above.

Third examination (November 30).—Semi-comatose. Cheyne-Stokes' respiration.

FIG. 2.

During apnœa left limbs flaccid, no trace of grasping phenomena. Right limbs retain their customary spasticity. Plantar response from left foot was sometimes plantar flexion and bunching of all toes and sometimes dorsiflexion of the hallux. Biphasic responses were also noticed.

Respiration became continuous during examination. The tonic resistance of left limbs and the grasping phenomena returned. At times when gently grasping the examiner's hand—during this and other examinations—the patient's left hand would actively and easily relinquish this grasp and travel down to his penis when about to micturate, or up to his face to rub his nose.

Fourth examination (December 5).—Semi-comatose, regular respiration. No response to presence of examiner. Left limbs show tonic innervation to stretching. No grasping movements to any mode of stimulation of the hand, but a rod placed in the curve of the semi-flexed fingers and pulled brusquely so as to stretch the finger flexors evokes a tonic contraction of these muscles of

moderate intensity. Thus, tonic innervation to stretch is the last remaining component of the grasping phenomena (fig. 1).

During the greater part of the period of observation the patient presented a catatonic condition of the left limbs. These retained for several minutes any posture imposed upon them, as is illustrated in fig. 2.

Post-mortem examination a few days after the last clinical examination revealed the presence of two cerebral tumours, one in the upper part of the left temporal lobe, the second in the left frontal lobe, near its mesial aspect and invading the corpus callosum. A large recent hæmorrhage was present in the latter. Histologically the tumours were gliomatous.

Summary.—A case of left frontal and temporal lobe tumours, presenting right hemiplegia, very slight left hemiparesis with grasping movements and tonic innervation of the left limbs. Under observation, the former gradually diminished and disappeared, tonic innervation of the flexors of the fingers persisting later. The grasping movements occurred in response to certain special forms of tactile stimulation of the hand, but not to other forms. Tonic innervation of the finger flexors was obtained only in response to stretch of these muscles and not to other forms of stimulus.

Case 5.—W. F. S., man aged 49. (National Hospital, 1933, Dr. S. A. K. Wilson.)

History.—Six months before admission to hospital had three generalized convulsions with loss of consciousness, within three weeks. Subsequently became progressively apathetic, confused and silent. Could not be got to answer questions. Complained of headache at time of onset. Progressive left hemiparesis, incontinence of urine.

First examination (December 19, 1932).—Conscious, motionless and silent. Will give his name slowly on request, but does not answer other questions. He follows the observers with his gaze.

Optic discs normal. Has useful vision. Fields cannot be tested.

Flaccid left hemiplegia, no voluntary movements can be elicited.

There is no definite right hemiparesis. He moves his right hand up to his nose or head, and back again to the bed from time to time, these movements being slow but otherwise normal and well co-ordinated. There is no visible delay in relaxation.

The tendon-jerks are of normal facility in the left limbs, slightly diminished in the right. Abdominal reflexes absent right and left. Plantar responses extensor right and left.

Sensation cannot be tested, but he makes grimaces when pricked on either side.

Right limbs.—The right arm lies semi-flexed on the bed. The muscles are very soft to palpation, but when the arm is passively extended at the elbow, biceps and supinator longus go into strong tonic contraction and stand up

beneath the skin in strong relief. It requires considerable force to extend the arm fully, the muscles remaining in tonic contraction until the limb is released (fig. 3).

Grasping movements.—The patient's hand is held round the wrist in front of him. He gazes fixedly at it. Repeated stroking movements are made with a pencil on the fold of skin between thumb and index. At each contact, these digits make a grasping movement to enclose it. If the process is repeated *without making actual contact with the skin*, the grasping movements occur as before, but cease immediately the patient's eyes are covered.

The hand is now released and the procedure repeated. The results are the same, except that the hand now moves actively towards the pencil before it reaches the interspace between thumb and index. These movements of the hand and arm through space cease at once when the patient's eyes are covered.

The examiner's fingers are now laid upon the patient's palm. His hand at once closes round them and enwraps them in a natural grasp. If the examiner keeps one or more fingers outside the grasp, the patient's fingers extend normally and make movements until they have enclosed the examiner's elusive fingers, and the hand now settles round the examiner's into a natural grasp. This grasp is gentle and remains so until an effort is made to pull the examiner's fingers away.

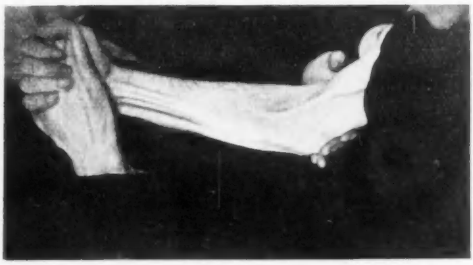

FIG. 3.

Tonic innervation.—If the examiner's fingers enclosed within the patient's grasp are pulled slowly away, especially if in such a direction as to stretch the patient's fingers and arm, the arm flexors and finger flexors go at once into strong tonic contraction, so that the patient's head and trunk can be pulled up free of the bed. Brusque orders to the patient to "let go" are without effect, though the patient appears to be trying to open his hand. As soon as the pull is released, the patient's hand opens naturally.

Tactile stimulation of the hand.—The patient's hand is laid palm up on the examiner's palm and held. Systematic testing with cotton-wool, pressure and pin-prick evoke no grasping movements and no trace of tonic innervation.

The empty grasp.—At times the patient opens his hand voluntarily as it

lies free on the bed. There is no delay in doing so. If his hand be closed by the examiner, and he be commanded brusquely to open it, at times he takes no notice, but occasionally he seems to appreciate what is required of him and opens his hand quickly and with ease. There is no tonic innervation in the opening of the empty fist.

The right leg lies semi-flexed on the bed. The muscles are flaccid to palpation, but passive extension of the knee or passive movements at the ankle at once evoke strong tonic resistance. The knee flexors stand up under the skin in strong relief, and so remain until the leg is laid down on the bed, when they relax at once and become soft again (fig. 4).

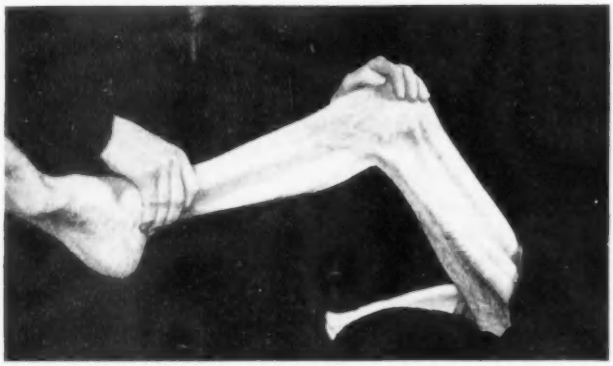

FIG. 4.

Stimulation of the sole by light or heavy stroking or pressure evokes no tonic contraction of plantar flexors, but only a feebly marked extensor response.

Examined during the same evening, the above results were all confirmed, but as soon as the patient fell asleep both grasping to contact and tonic innervation disappeared.

Examination on December 20: State of consciousness as before.

Strong tonic resistance to extension of the elbow or of the knee.

Voluntary movements of arm normal in rate.

Grasping to pencil introduced between finger and thumb as before, both to vision and to touch.

Systematic sensory examination of palm as described above evoked no grasping movements.

Tonic innervation of arm and leg and of grasping muscles to stretch as before.

Examination on January 16, 1933: The patient is now emaciated and feeble, but remains in the same condition of conscious immobility. He is totally irresponsive to questions and orders, and does not fix examiner with his gaze. There are no grasping movements when the palm is stroked, or when the examiner's hand is laid within the patient's.

If the examiner's index finger is placed in the hook of the patient's semi-flexed fingers and brusquely pushed into the digits so as to extend them, they at once go into strong tonic flexion and retain the examiner's finger as long as this is pulled away from the patient.

Examination on January 17, 1933: Patient less apathetic. He fixes examiner with his gaze and at once offers his hand, moving this up off the bed towards the examiner who stands beside the bed. He does not answer questions, or carry out simple orders, such as "Shut your eyes," etc.

He encloses the examiner's hand when this is placed on his in a gentle grasp.

Tonic innervation of arm and of grasping muscles to stretch as before, though not so strong.

Sensory testing of hand with cotton-wool, pressure, &c., evokes no trace of grasping, but immediately afterwards the examiner's hand is placed on the patient's and is again enclosed in a grasp. If a finger eludes the grasp, the fingers open and feel about for the missing finger until it is found, when it is enclosed in a natural grasp.

The patient died on January 26, 1933. Post-mortem examination revealed the presence of a large meningioma of the right frontal region, surrounded except at its point of attachment by a thin shell of frontal lobe. The lobe was greatly expanded by the tumour and extended across the mid-line, compressing the opposite frontal lobe.

Summary.—A frontal tumour producing severe hemiplegia on the opposite side with slight hemiparesis on the side of the lesion. The hand and arm on this side displayed grasping movements to certain tactile stimuli applied to the hand, and occasionally the patient offered his hand to the examiner directly he caught sight of him. Tonic innervation was present and could be elicited by stretch alone. It survived after grasping movements had ceased to occur.

Case 6.—F. I., a woman, aged 61. (National Hospital, 1931, Dr. Gordon Holmes.)

History: Three months before admission to hospital she suddenly became unable to express herself clearly or to write a coherent letter. She became progressively more silent and apathetic, and within a week was doubly incontinent, confused and unable to construct even a simple sentence. She allowed food put into her mouth to remain there unchewed and unswallowed.

On admission she was emaciated, motionless, except for following moving persons with her gaze, and unable to answer questions. She exhibited marked catatonia, her limbs retaining postures passively imposed upon them for a minute or longer. She was incontinent of urine and fæces.

Examination revealed bilateral papillœdema, tonic resistance to passive movement of all four limbs, tendon-jerks of normal facility and equal on the

two sides. The abdominal reflexes were present bilaterally, the plantar responses bilaterally flexor.

Her mental state was such that no co-operation with the examiner was possible. She died within a week of admission to hospital and autopsy revealed a small glioma in the left parietal lobe, and a second one on the mesial aspect of the right frontal lobe invading the corpus callosum.

Grasping movements.—Touching and stroking of the palmar aspect of hand and fingers elicited no trace of grasping movement. Deep pressure and pin-prick evoked no response of any kind.

Tonic innervation.—The patient's hand (right or left) is turned palm up, and the slightly flexed digits are brusquely pressed downwards, i.e., are extended. At once they flex round the examiner's pressing finger and grasp this firmly and for as long as extension (i.e., stretching of the flexors) is kept up. If the examiner now pulls his finger away from the patient so as to extend both arm and digits, this tonic grasp increases in force.

If the patient's hand is fully flexed at the wrist before this procedure is adopted—that is, if the flexors of the fingers are made as lax as possible—then extending the fingers in the manner described elicits either no response or a feeble one.

If now the patient's hand be fully extended at the wrist, then pushing down the fingers (that is, still further stretching the flexors of the digits) evokes a strong tonic flexion round the examiner's pressing index finger.

The legs present the same tonic resistance to passive movement as the arm, but no abnormal response is obtained from the toes by any mode of stimulation of the sole.

Summary.—A case illustrating the last stage in the dissolution of the grasping and tonic phenomena, the former having completely disappeared—if we may assume that they were at one time present.

Chapter II.—Summary and Analysis of Clinical Observations.

Of the eight cases which form the basis of the present paper, six have been recorded in some detail. In all there was evidence of the presence of a lesion involving the frontal lobe, and in all but one (Case 6) there were also signs of an associated pyramidal lesion. In this one, pyramidal signs never developed, and in the remainder it is probable in some instances at least that grasping and tonic phenomena preceded the appearance of these.

When found, pyramidal signs are such as to indicate a relatively slight impairment of function in this system of neurones. Apraxia was not observed in any of our cases.

Not every one of our cases has shown the full range of grasping and tonic phenomena, and those that have done so have not shown it

throughout the whole period of observation. These phenomena develop gradually, reach full development, and then with the progress of the illness they undergo a progressive restriction of range, accompanied by an increasing impairment of consciousness, until finally with the onset of stupor and semi-consciousness, nothing remains but tonic innervation elicited by stretching the muscles involved. Under the conditions of a progressive dissolution of nervous function this component has been the last to disappear.

Of all our cases we may make this generalization, namely, that all showed some unequivocal signs of impairment of psychical function—some signs of dissolution of function in the highest cerebral centres, and we believe this to be an invariable concomitant of grasping movements, and probably also of tonic innervation.

We may most concisely summarize our observations under four clearly marked grades of development which in our experience they have presented.

I.—The grade of evolution, seen in patients who are relatively little reduced psychically and who can co-operate in their examination.

In these we have found (1) that when the patient's attention is directed elsewhere, his affected hand closes gently round the examiner's hand when this is slipped into it between thumb and index finger, or when the patient's palm is stroked by the examiner's fingers. When the examiner's fingers are insinuated into the palm from the ulnar side, there is often no trace of grasping.

(2) When the patient is asked to turn his palm upwards and this is then tested systematically—within his sight—with cotton-wool, stroking, pressure and pin-prick, there is no trace of grasping movement.

(3) When the patient is asked to clench his fist, either with the hand empty, or over some object, such as a fountain pen, he can at request open the hand with perfect ease and without delay. Any object that the hand may contain in its grasp drops out freely when it is opened in this way.

(4) Pressure exerted against the palmar aspect of the digits so as to stretch the flexor muscles at once leads to the development of tonic contraction in these muscles, which persists until the fingers are released and stretch of these muscles ceases. This may be elicited either by trying to pull some object out of the patient's grasp, or by inserting a finger or a wooden rod in the crook formed by the semi-flexed fingers and then pulling away.

(5) The patient can easily prevent the occurrence of grasping movements, but once tonic innervation is produced by stretch the patient has no control over it, and only after great and repeated effort can he extend the fingers and release them from the pull.

(6) The patient's hand does not wander towards the examiner's hand when this is offered, nor does it follow this after it has been withdrawn. Tonic innervation appears to be first of the phenomena under consideration to develop.

II.—The grade of full development—seen in patients whose state of consciousness and intelligence is still sufficient to allow of visual fixation and of some degree of attention.

In these we have found (1) a grasping of the examiner's hand, when this is within the patient's field of vision, and is offered to the patient or laid beside his affected hand.

(2) In these circumstances the patient's hand and arm move towards the examiner's hand, and follows it when it is withdrawn, whether contact is made with the hand or not.

Both (1) and (2) cease immediately if the patient's eyes are covered.

(3) A grasping of the examiner's hand when this is laid upon the patient's palm, and this whether the patient's eyes be open or shut.

In all three instances the grasping movement is deliberate and very skillfully co-ordinated so as to secure, not merely a hold, but a natural and complete grasp. If means be taken to obstruct this end, the grasping fingers continue to move round the hand until this has been completely and comfortably obtained.

Further in all these instances the patient can prevent the occurrence of the movements when asked to do so. If his attention wanders they restart. This form of grasping is neither "forced" nor involuntary in the strict meaning of these words.

(4) When the patient's open palm is repeatedly touched by the examiner's finger, each touch is followed by a grasping movement of the digits, and when the patient can see the finger his arm may move towards it and may follow it on its retreat. Similarly if in these repeated movements actual contact with the patient's hand is avoided, the latter still makes the movements described so long as the examiner's finger is visible to the patient. If his eyes be covered there is no response except to contact, and then nothing but finger movements.

(5) Systematic sensory testing of the patient's palm as described above evokes no grasping.

(6) Tonic innervation is elicited as described above and only thus.

(7) The patient can open his empty fist with perfect ease.

III.—Seen in patients who are completely apathetic or mildly stuporose and have ceased to be able to maintain attention—the first grade of dissolution of grasping phenomena.

(1) and (2) as described under (II) have disappeared.

(3) Present as described above.

(4) Grasping movements of fingers to contact. No movement of arm and no response without contact.

(5) and (6) as described above.

(7) Can open clenched fist easily when able to understand the order.

At this stage catatonia may be present.

IV.—Final grade of dissolution—seen in patients who are semi-conscious and cannot be roused to attention.

No grasping movements of any kind. But tonic innervation to stretch persists, though diminished in force.

The observations thus briefly summarized indicate that we are dealing with phenomena of two distinct orders: (1) *a grasping movement* of the fingers, sometimes accompanied by a movement of the whole hand and arm through space, which is highly co-ordinated and capable of a degree of adaptation that unmistakably reveals the activity of the cerebral cortex. The stimulus that evokes the grasping movement of the fingers may be either visual or tactile, but no movement of the limb through space occurs unless the patient can see and fix with his eyes the stimulating object. When the patient's eyes are covered nothing remains but a grasping movement of the stationary hand to tactile stimulation.

Not all visual or tactile stimuli are adequate. For a visual stimulus to be effective it must be such as to make grasping a natural response; the patient must be "offered" the examiner's hand, or this must be laid beside his own. Similarly with tactile stimulation of the hand, moving contacts with the palm such as are made by an offered hand are effective, but systematic sensory examination of the skin of the hand carried out in the manner described above and of an order not naturally productive of a grasping response fails to yield such a response. Finally the grasping movements of hand and the movement of the limb through space can both be prevented by the patient, and are to this extent within his control.

(2) *A tonic contraction* of muscles elicited solely by stretching, and resembling other known tonic reflexes in persisting for as long as the adequate stimulus is kept up. This contraction is wholly outside the patient's voluntary control. It is uniform in character and does not show the highly delicate adaptation to changing circumstances shown by the grasping movements. Unlike the latter it may occur in the semi-conscious patient.

With the question of the localization of the cerebral lesion necessary to allow of the development of the phenomena described we are not specially concerned in this paper. Our own cases, like those of our predecessors, have probably all had a lesion involving the frontal lobe, particularly the frontal and intermediate precentral regions, and the experimental studies in monkeys that are considered in a later chapter confirm the results of clinico-pathological study in this respect. We will make no other comment than to say that in every case, human and animal, in which grasping phenomena or tonic innervation have been seen, the region of the cortical origin of the fronto-pontine path seems to have been involved. Therefore, impairment of function of this neurone system seems to allow the release of the grasping phenomena.

It is true that Bucy [3] has recently recorded two cases of intracranial tumour in which the primary lesion was remote from the frontal lobe. But in each the brain was subjected to severe pressure and internal hydrocephalus was present. The first, and only adequately described case, was one of occipital glioma. Grasping phenomena were a very late development and there was also bilateral external rectus weakness. In these circumstances no neurologist would regard either of these signs as of localizing value.

They merely indicate that under conditions of extreme intracranial hypertension impairment of nervous function spreads far from the site of the initial lesion, a fact with which all neurologists are familiar. They do not conflict with the well-established observation that the lesion underlying grasping phenomena is in the frontal lobe, whether this lesion be a softening or tumour in this situation, or a compression resulting from a tumour elsewhere within the skull.

CHAPTER III.—THE GRASPING MOVEMENTS CONSIDERED AS VOLITIONAL MOVEMENTS.

Anyone who has carefully observed the grasping movements of the hands that we have described, who has seen the high degree of purpose and skill they display in achieving their object, their perseverance until

this has been attained, their subordination to the patient's will—when he can be induced to exert this, and also the extremely specialized stimuli which are necessary to evoke them receives the ineradicable impression that they are voluntary movements. There is an adaptability behind them that allows of no other interpretation, and their dwindling and disappearance with the waning of the patient's consciousness seems to confirm this impression.

There can be no doubt that they do not belong to the class of fixed, inborn reflex reactions of the kind that the researches of Sherrington and other physiologists have made us familiar with, nor with comparable reactions studied clinically in man. And if the identification with these is the basis of the title of "grasp reflex" that has been conferred upon the movements with which we are concerned, then the interpretation is clearly erroneous.

We may say of them that they are volitional movements occurring in individuals whom disease of the brain has reduced to a lower level of internal evolution in respect of their highest motor functions. One of the most striking single instances of the volitional nature of grasping movements was afforded by Case 5. At a period when, for some weeks, he had betrayed no initiative of any kind, and lay speechless and motionless and with fixed stare on his bed, he was examined on many occasions. On one such occasion the examiner approached the bed and was caught by the gaze of the patient, who at once slowly raised his right hand from the bed and offered it to the examiner, obviously to be grasped.

To grasp an object felt by the hand, and then to grasp an object seen, are the first and second stages respectively in the individual's acquired use of the hand. They represent the most deeply organized and therefore most automatic activities of the hand, and their prominence in the circumstances we are considering represents the result of "a "taking to pieces" in the order from the least organized, from the most complex and most voluntary, towards the most organized, most simple and most automatic" (Croonian Lectures, Hughlings Jackson). To label these movements "automatic" in contrast to "voluntary," is merely a matter of terminology, for "most automatic" is nothing but "least voluntary."

It might be urged, however, that to express the situation thus is to do little more than to restate the facts of observation, and that there is nothing in these movements which, under a liberal definition of the term, might not be called reflex. Further, it might be maintained that

they are in fact characteristic examples of that group of reactions which Pavlov has called "acquired" or "conditioned" reflexes, and that they differ from the fixed, inborn reflex reactions of the decorticate nervous system only in degree of refinement and not in essential quality. To be precise, the features in which the movements we are considering differ from the latter may be stated as follows: (1) They may be elicited from either of two widely separated receptive fields, visual and tactile; (2) within these fields the adequate stimuli have become highly specialized. An object is not grasped every time it is seen, nor every time it is felt by the hand. It is grasped only when the stimulus is such that grasping is what the individual's experience has taught him to be an appropriate and satisfying response; (3) the response is a far more complex and flexible one than is that of the inborn reflex; and (4) the response can be prevented from occurring by the patient, even when the adequate stimulus has been delivered.

These are indeed the features which Pavlov has described in the reactions he has studied, but we may equally well say of them that they are the characteristic features of cerebral cortical activity in voluntary movement, namely, "reaction to difference, flexibility of response to the same stimuli and habituation to stimuli" [4].

In a recently published critical analysis, Denny Brown, whom we have just cited, has subjected to a very searching criticism the theoretical deductions which Pavlov has drawn from his observations, in particular his use of the terms "reflex" and "inhibition." He points out that to expand the conception of reflex action to include what he prefers to speak of as "conditioned responses" is to make this mean no more than response to stimulus. But this quality may be said to belong to all possible movements. In short, we can speak of these responses as reflex only by depriving this term, and the conception it embodies, of all precise meaning.

This criticism would apply with equal force to a description of the grasping movements as reflex. We add nothing to our knowledge or understanding of them by so naming them, while we confuse the use of what has been, and should remain, a term of precision.

Therefore, we prefer to speak of them as volitional movements. Finally, a word as to nomenclature. These movements are not "forced" in the sense that this description is customarily employed in neurology; as, for example, in respect of the forced movements of some vestibular lesions. Nor are they forced in any figurative sense. Further, the word "groping" implies blindness or darkness, and cannot apply to

movements directed by vision, as we have seen the movements so named both necessarily and invariably are. There is little, therefore, to commend the expression "forced grasping and groping," either as scientific, or as usefully descriptive.

Catatonia and slowness of movement.—If the subjects who present grasping movements and tonic innervation be observed over a considerable period and until their final lapse into coma before death, it is seen that at a certain level of dissolution of cortical function they become completely apathetic and stuporose, lying silent and motionless in bed, passing urine and fæces under them, and betraying little sign of consciousness beyond occasional movements of the eyes when they see someone passing, or slow movements of the hand and arm to rub nose or face. At this stage in their course catatonia is commonly present, and their movements become extremely slow. It is possible to place a limb in some posture where it remains motionless for one or more minutes.

All these phenomena, grasping movements, catatonia and slowness of movement, express a severe degree of reduction of cerebral function towards the least voluntary and most automatic, and to this category of low-grade volitional activities they all equally belong.

Catatonia was most strikingly observed in Case 4 (H. B.), as is illustrated in fig. 2, in which he is seen to be maintaining positions, passively imposed, of both arm and leg.

Chapter IV.—Tonic Innervation Considered as a Proprioceptive Reflex Reaction.

When the Babinski plantar response was first brought to notice, its great practical usefulness as an instrument in diagnosis for long obscured its significance as a fraction of a larger reflex reaction of the lower limb to stimuli of a certain quality. Similarly, the clinical importance of tonic grasping as a "localizing" symptom has distracted attention from the fact that it is but the local expression of a more widespread disorder of neuromuscular activity. It may be said, in fact, that the grasping is fortuitous and does not necessarily provide any clue to the physiological significance of tonic innervation.

It is particularly in the case of the rarely observed tonic innervation of the plantar-flexor muscles of the toes that the term "grasp reflex" is most obviously inadequate and fanciful. (Brain and Curran [2].)

The mechanical arrangements of the foot are such that when all the toes are plantar-flexed they bunch together, and are then capable of

holding any appropriately shaped object inserted between them, or put between hallux and sole by the observer. But to suppose that this event has anything to do with grasping in a physiological sense is no more than one of those unconvincing phylogenetic fantasies that see in almost every anomaly of movement the hypothetical activities of our "arboreal ancestors" [16].

We shall therefore adhere to the term "tonic innervation," which lays no undue emphasis upon local phenomena and embodies no hypothetical implications.

The sole adequate stimulus for the production of tonic innervation appears to be stretch of the muscles concerned, a proprioceptive stimulus of the order constantly found in association with tonic reflexes.

Tactile stimulation of the hand we have found to be uniformly ineffective, a result that was to be anticipated in the case of a tonic reflex.

Not only is stimulation of the palm by the patient's own fingers inadequate, but even that delivered by some object held in the grasp is without effect unless this is pulled in such a way as to produce stretch.

While in most of the recorded examples of tonic innervation the cutaneous sensibility of the affected hand has been either normal or but slightly impaired, there are two cases in the literature in which this was not so.

Thus, of Schuster and Pinéas' Case 10, it is said that "Sensibilität für alle Qualitäten ausser Lagegefuhl auf den linke Seite herabgesetzt," while Rhein [11] has recorded in detail a case in which apraxia and tonic innervation (grasping movements not mentioned) occurred in a blind patient whose affected arm was totally deprived of all modes of sensibility.

Too great weight may easily be placed upon cases cited from the literature, but in so far as these two have any bearing upon the question of tactile stimulation of the hand as a stimulus to tonic innervation they are in harmony with our own observations.

The uniformity shown by this reaction in respect of stimulus, receptive field, response and independence of voluntary control shows it to belong to the group of tonic reflexes of the segmental nervous system with which we are familiar both in animals and in man.

Cortical activity plays no part in it, and it stands upon a lower physiological plane than do the grasping movements, the extinction of which it survives.

To passive manipulation, the curious hypertonus of the muscles

engaged in tonic innervation differs somewhat from the spasticity of pyramidal lesions. It persists unabated throughout the range of passive movement, and lacks the clasp-knife quality of spasticity, and while the latter remained unchanged throughout the apnœic period in one of our cases, tonic innervation disappeared constantly with apnœa.

Investigation by electrical and myographic methods, or by novocain de-afferentation [15], may throw further light upon its nature and its relation to spasticity.

In none of our cases was tonic innervation of the plantar-flexor muscles observed.

One point remains for mention. It would appear from some descriptions [17] that tonic innervation may ensue upon voluntary contractions and cause delay in relaxation in circumstances where it is not clear that stretch has been operative as a stimulus. Unfortunately for our purpose, the early writers attributed no significance to this factor, did not notice its presence, and in their descriptions give us no idea as to whether or not it was excluded. Thus, the statement that a patient could not open his hand means nothing unless we know the following details: namely, whether stretch was operative, whether the patient understood what was required, whether apraxia of the hand was present, and whether the limb was spastic or not.

Observations repeated many times in our series of cases constantly failed to detect the occurrence of any delay in muscular relaxation when care was taken not to subject the muscles to stretch.

Chapter V.—The experimentally produced "Grasp Reflex" and its Relation to the Phenomena herein described.

The physiologist can determine so many of the conditions of his experiment and can subject his material to such exact study that he undertakes the investigation of this problem with advantages denied to the clinical observer.

This is illustrated by the precision with which experimental study has defined the cerebral lesion necessary to allow of the development of tonic innervation. Unfortunately, the special aspect of the problem with which we are concerned, namely, the analysis of the essential components of grasping phenomena, has received no illumination from this source, except what we may derive by inference.

In the adult macacus monkey, Richter and Hines [9] found that ablation of the intermediate precentral cortex ("premotor area,"

Brodmann's area 6) is followed constantly by the development of a "grasp reflex." This consists in tonic grasping when the animal is hung by one hand from a horizontal bar, and may be elicited for as long as twenty-one days after operation. The reaction is crossed, but bilateral ablation of the relevant cortical region gives a stronger response in each hand than can be obtained in either by unilateral ablation. Nothing is said in Richter and Hines' brief note about grasping or groping *movements*. It is impossible to say whether in these experiments tactile stimulation of the hand or stretching of the finger flexors provided the adequate stimulus. The method of conducting the observation, namely, hooking the animal's digits over a bar and then releasing the animal, certainly suggests that stretch may have been the essential stimulus.

Richter [6] has also examined the "grasp reflex" in newborn monkeys, and finds that these animals hang for many seconds from a horizontal bar by one hand when this is hooked over the bar in the manner described above. Here also the crucial question of nature of the stimulus is not answered, and there is no mention of grasping or groping movements.

Richter and Paterson [7, 8] have found that appropriate doses of bulbocapnine will produce the same phenomenon in monkeys, the maximum period of hanging being longer in immature than in adult animals. Here, again, nothing is said of any grasping movements, and one is left by these three series of experiments with the impression that all that has been seen is tonic innervation elicited by stretch.

More favourable opportunities of deciding the question appear to have been afforded by some experiments of Fulton, Jacobsen and Kennard [5]. They performed ablation experiments similar to those of Richter and Hines, and their animals showed what have been called "grasping and groping."

Unfortunately these observers have adopted the current hypotheses and nomenclature without question and have made no attempt to study the details of stimulation essential to elicit the phenomena they observed. After bilateral ablation of the frontal and premotor (Brodmann's area 6) cortex they noted (1) that the animal slowly reached for and grasped any object brought within its field of vision; (2) that light stroking of the palmar surface of hand and digits evoked vigorous grasping movements; (3) that a tonic contraction ensued upon this grasp which was of sufficient strength to support the animal's weight for several seconds; (4) that vigorous "grasping" of the foot was seen in one of their two experiments.

We do not learn from these experiments how the tonic grasp was elicited, though it appears probable that here also, hanging the animal from a bar was the procedure adopted. This at once involves the possibility of proprioceptive stimulation. As for the movements of grasping and pursuit, they confirm that vision alone is adequate to elicit them; as to whether they were volitional or reflex we are left in the dark.

If we are justified in drawing any conclusions—other than anatomical—from these physiological experiments, they would be that the observations of Richter and his co-workers indicate that tonic innervation may occur alone without grasping movements, and that stretch is the stimulus necessary to evoke it.

General Summary.

The phenomena included under the various headings of "tonic innervation," "the grasp reflex," "forced grasping and groping," and the like are found to be capable of analysis into two distinct and separable components, the one volitional and the other reflex.

(1) The volitional component includes grasping *movements* of the fingers, with which movements of the hand and arm through space are sometimes associated. The stimuli to which they are responses are visual and tactile. Often both are operative, and it is only when the patient has the use of vision that movements of the limb as a whole occur. Tactile stimuli alone evoke nothing but grasping movements of the fingers, the hand and arm remaining still.

On the other hand, visual stimuli acting alone are adequate to evoke the full range of movements.

Only those visual and tactile stimuli are effective to which grasping is a natural and satisfying response. The movements are under the patient's control in that he can prevent their occurrence. They show a delicacy of co-ordination and a capacity for adjustment to changing circumstances that, with the highly specialized stimuli necessary to elicit them, reveal the participation of the cerebral cortex in their production. In these qualities they differ from the fixed, inborn reflexes of the decorticate nervous system, and the term reflex cannot be rightly applied to them. They are in fact volitional movements occurring in individuals whose psychomotor functions have been reduced by disease of the brain to the production of the "least voluntary" and "most automatic" movements. They are not "forced," or involuntary. They occur only in the conscious patient, and disappear with the development of stupor or coma.

(2) The reflex component consists in the tonic innervation of muscles when, and only when, these are subjected to the proprioceptive stimulus of stretch. Tactile or visual stimuli are not adequate to produce it. It may occur in the conscious or in the unconscious patient, and it is entirely outside the patient's control. The stimulus and the response are fixed and uniform and indicate that we are dealing with a true tonic reflex of the segmental nervous system, and one in which the cerebral cortex plays no part.

Tonic innervation of the grasping movement occurs only when the grasping muscles are subjected to stretch, as when the attempt is made to extract some object held in the grasping hand. When all grasping movements have disappeared with the waning of consciousness, tonic innervation persists and can be elicited by sudden passive extension of the fingers.

The tonic grasp is only the local expression of a more widespread mode of innervation of the muscles, and is in this sense a fortuitous phenomenon that does not necessarily provide any clue as to the origin or significance of tonic innervation. For this reason the term "grasp reflex" is undesirable as conveying a false emphasis upon what is no more than a local manifestation. This is particularly the case when the term is applied to tonic innervation of the plantar-flexors of the foot described by some writers which, even when dressed in the language of metaphor, is not obviously a grasp.

The experimental studies of grasping phenomena in so far as they throw any light upon the problem considered in this paper, suggest that tonic innervation is a true proprioceptive tonic reflex, of which grasping movements are an uncertain concomitant.

The lesions which allow of the development of grasping movements and of tonic innervation appear to be situated in the frontal lobe, anterior to the excitable motor cortex and adjacent to the longitudinal fissure. Experimental studies on monkeys indicate that the intermediate precentral region ("premotor area," Brodmann's area 6) is the precise localization of the essential lesion.

In respect of nomenclature, the two separate components described should be dealt with separately. For the volitional component no term more precise than that of "grasping movements" is necessary or justified, since these movements are neither reflex, nor "forced." For the reflex component, "tonic innervation" remains the most suitable term in the present state of knowledge.

To those members of the Medical Staff of the National Hospital

who have allowed us to examine and record their cases, we express our thanks.

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THE RATE OF CONDUCTION AND REFRACTORY PERIOD OF THE HUMAN SENSORY NEURONE.

BY F. GOLLA AND S. ANTONOVITCH.

(*From the Central Pathological Laboratory of the London County Mental Hospitals.*)

SUCH investigations as have been conducted on the physiology of the nervous system of the intact human being have been almost entirely confined to the efferent portion of the nervous system, or at any rate to such experiments as may be checked by the muscular response.

On the sensory side we have, however, means of judging the effect of excitations by our sensory impressions which are intrinsically more delicate than the investigation of motor responses and allow absolutely normal physiological conditions instead of the extensive dissection and exposure necessary to allow records of sensory processes to be obtained by amplification of the electrical response. It is hoped that the experiments to be described will permit the evaluation of the effects of drugs and states of chemical equilibrium under conditions which could not possibly be so favourable in animal experiments.

Method.—It was necessary to deliver two successive stimuli at variable intervals. The stimuli had to be of such short duration that the time through which they acted was negligible and the time intervals determinable within 0·1 sigma. A Lucas pendulum was used, so adjusted as to allow determinations of the time relations within 0·1 sigma. The velocity of the pendulum when used in conjunction with the electrical contacts employed in these experiments was determined both before and after a series of experiments, using a string galvanometer as recorder in the usual manner. The variations of the velocity were found to be negligible. The type of key originally designed by Lucas for break shocks was modified in such a way that only an instantaneous closure of the circuit occurred when the pendulum struck the steel pin of the key (fig. 1). To effect this the striker of the pendulum was connected with one arm of the circuit, the other connection being made to the knife edge on which the steel pin rests. The circuit is closed for the very brief interval of time elapsing between the moment when the striker first makes contact with the steel pin, and that when the pin is

pushed by the striker from the knife edge. The time during which the current is closed is negligible for the purposes of these experiments, being less than 0·01 sigma. The very small intervals during which the circuits were closed were found not to be absolutely equal, and this inequality manifested itself as a difference in intensity of the stimulus. The stimuli led off from the two keys had therefore to be equalized when the electrodes were applied by the use of non-inductive resistances. A battery of accumulators of 120 volts and a suitable potentiometer completed the circuit.

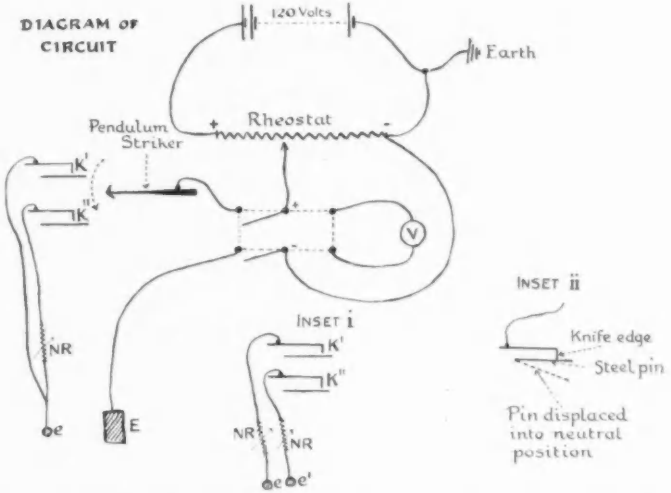

FIG. 1.—The Lucas Pendulum is represented diagrammatically as two keys (K', K'') and the striker K'' is movable along a graduated scale, so that the interval between the two stimuli can be varied and accurately determined. In the diagram both stimuli would be applied at the same point on the skin. Inset i shows the arrangement by which two distant points can be stimulated successively. E = indifferent electrode. NR = non-inductive resistance. V = voltmeter. Inset ii shows a key in detail.

The electrodes used were at first similar to the silver non-polarizable electrode of Bourguignon, the neutral electrode being about 8 sq. cm. in size and the stimulating electrodes having each a surface of one square centimetre. It was subsequently found that equally good results were obtained with zinc electrodes. When the successive stimuli were delivered at one spot a common electrode was used.

The choice of a suitable subject must be a matter of great importance if the results here presented are to be verified. These investigations having been begun with one of the authors as a subject, we have

continued to depend mainly on the same subject throughout these experiments. All the conclusions, were, however, verified on a laboratory assistant who proved to be a good and objectively minded witness. Two other youths proved, however, to be useless as subjects, at any rate without more training than we had leisure to give. So far as it was possible for us to judge, their chief faults were inattention and a tendency to guess when not sure.

From introspection it is possible in some degree to define the state of mind necessary for a successful result as one in which in describing a sensation all qualities other than the particular one studied for the purpose of the experiment are ignored. Thus, in judging summation, nothing but the magnitude of the sensation must be considered and spatial and temporal references must be neglected. In judging the presence or absence of multiplicity of the stimulus, the temporal reference must be considered, to the exclusion of magnitude and localization. The fluctuating qualities of the local sign present a very puzzling and disturbing element. It would appear that within a small area underlying the stimulating electrode there is a continuous fluctuation of the skin spot to which the stimulus is assigned. Any two stimuli following each other at a sufficiently brief interval are invariably assigned to the same spot, but after an interval of a minute a second pair may appear to affect a spot which is felt to be at some little distance away, and the second pair appears to possess a different affective tone to the first. It is doubtful how far these variations are due to alterations of skin conductivity of the same nature as the so-called psychogalvanic reflex, which is conditioned by concomitant alterations of the affective tone. Whatever be the interpretation of these fluctuations of affective tone and local sign, their occurrence renders it necessary for the stimuli which are being compared to be delivered in pairs and for the observer to pronounce judgment on each pair without reference to any preceding pair. This method of pairing obviously increases very greatly the number of stimuli that have to be administered, and makes great demands on the endurance of the subject. It was found that experiments were greatly facilitated when a warning preceded the delivery of the stimulus, and this was very conveniently rendered by the noise of setting the keys before each swing of the pendulum. These movements had to be performed with a strict observance of tempo—any irregularities in the time interval between the warning signal and the stimulus caused disturbance of the subject's attention.

The refractory period of the afferent nerve.—In the first instance

this was determined in the ulnar nerve. The stimulating electrode connected with the keys K′ and K″ was firmly bound on to the skin overlying the nerve at the ulnar groove by sticking-plaster. The large neutral electrode was applied to the lower dorsal region. In other experiments the supraorbital nerve was selected at its point of emergence from the supraorbital groove, and in another series the musculocutaneous nerve as it winds round the head of the fibula. From all these three points identical results were obtained. There is, of course, no reason why stimulation of the cutaneous nerve at any place should not yield equally satisfactory results, and this was found to be roughly true, but the points enumerated had been selected in the course of some earlier experiments directed to other purposes and were adhered to as a matter of convenience. The voltage was adjusted to give two plainly perceptible stimuli when the keys K′ and K″ were separately closed by the pendulum. The stimuli were then adjusted so as to be of equal intensity by means of the non-inductive resistances. This was necessary, as intrinsic differences in the tension of the springs of the two keys by causing minute differences in the time of closure rarely gave an equal strength of stimulus from either circuit. When the stimuli were judged to be of equal intensity, the voltage was lowered till both stimuli were just a little above the threshold of sensibility. The exact intensity of stimulus for optimum results can only be settled by the experience of the subject, in view of the impossibility of gauging the skin resistance to the constant current owing to polarization. The moving key K″ is now adjusted so that a double stimulus is given practically simultaneously, and this is compared with a single stimulus from one or other of the two circuits. So long as the second stimulus falls within the total refractory period following the first, the double stimulus will be judged to cause a sensation equal in intensity to that of the single stimulus. When the key K″ has been moved to a point at which the second stimulus falls in the partial refractory period of the first, it will be noted that the double stimulus effect, owing to summation, is now slightly greater than the effect of the single stimulus. This point, marking the end of the period of the total refractory state, can be determined within 0·1 sigma. As the second stimulus is moved further and further through the partial refractory period, a progressive increase of the strength of the double stimulus effect is noted, and the increase can be registered step by step within limits of 0·5 sigma. Finally, a point of maximum sensation is reached, and for some time the double stimulus continues to give effects of uniform intensity while the second

stimulus is being moved further and further from the first. When an interval of about 12 sigma between the stimuli is reached, the intensity of the sensation tends to diminish and, as will be seen later, this point corresponds with the sensation of reduplication. This diminution of intensity is, however, very difficult to localize in time with the degree of accuracy corresponding to that of the points for the refractory period.

The total and partial refractory periods here determined are undoubtedly those of the afferent nerve itself. They correspond closely to the total and partial refractory periods observed when the ulnar nerve is stimulated by somewhat stronger currents and the resultant contraction of the abductor minimi digiti is mechanically recorded

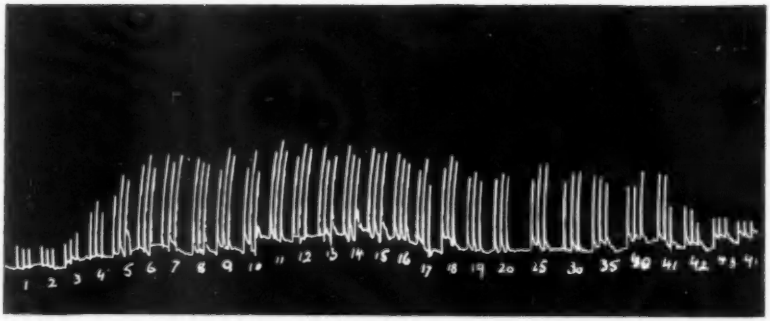

FIG. 2.—Tracing of movements of little finger in response to stimulation of ulnar nerve at olecranon. Figures indicate scale-reading on Lucas Pendulum. Three contractions are recorded for each reading.

(fig. 2). It will be observed that the total refractory period is appreciably more prolonged in the motor nerve, but it must be borne in mind that the mechanical record offers a far coarser testimony than that of sensation.

In the tracing the contraction of abductor minimi digiti is recorded to stimulation of the ulnar nerve at the olecranon by pairs of stimuli with lengthening interval between the stimuli.

The contractions are recorded in groups of three for each stimulus interval. The figures refer to the instrumental scale. Summation begins with a stimulation interval of 2·5 sigma, which represents the length of the total refractory period. It reaches a maximum at about 5 sigma stimulus interval which represents the end of the partial refractory period. Summation begins to subside at a stimulus interval of about 13 sigma.

The absence of any objective value for the intensity of sensation makes it impossible to represent summation graphically. The following table represents the time relations of the total and partial refractory periods.

	Stimulation interval	Sensation of double stimulus
Total refractory period ..	0·1 sigma	Same as single
	0·2 ,,	,, ,, ,,
	0·5 ,,	,, ,, ,,
	1·0 ,,	,, ,, ,,
	1·5 ,,	,, ,, ,,
	1·6 ,,	,, ,, ,,
	1·7 ,,	,, ,, ,,
Partial refractory period ..	1·8 ,,	Greater than single
	2·5 ,,	Greater than at 1·8 sigma
	3·5 ,,	Greater than at 2·5 sigma
	4·0 ,,	Same as at 3·5 sigma
	6·0 ,,	Same as at 3·5 sigma
	8·0 ,,	Same as at 3·5 sigma
Onset of sensation of reduplication	12·0 ,,	Less than at 3·5 sigma but greater than single

Another method of demonstrating the total refractory period is to diminish the intensity of the two successive stimuli till they are each just below the threshold of sensation. If the two stimuli be then delivered together within the total refractory period no sensation will occur, and the time value for the onset of summation at the expiration of the total refractory period can be determined very sharply by noting the first appearance of sensation. The curve of summation is, however, less easily followed when working with such minimal sensations.

The values obtained for the total and partial refractory periods and for summation apply obviously to the sensory nerve in the first instance. There is, however, no evidence that will allow us to decide how far the time values assigned to the refractory period and summation apply to superior neurones in the nervous system. It is by no means inconceivable that the greater or lesser impulses conditioned by the refractory period of the lower sensory neurone are translated by superior neurones possessing entirely different refractory and summation periods. Should this be so, however, the universal application of the all or none law to the neurones of the central nervous system would then make it necessary to postulate that the larger and smaller responses conditioned by the total and partial refractory periods of the lower motor neurone are transformed into repetitive discharges of varying frequency when the impulse is passed on to the superior neurone, possessing a different refractory period.

The reduplication of sensation.—When two successive stimuli are separated by an interval of 10·6 sigma the sensation is found to have a multiple or manifold character as if resolvable into two or more separate

stimuli. We have termed this new sensation "reduplication," but it should be understood that introspective analysis does not allow the subject to state clearly that the new sensation is composed of two separate sensations. All that he can say is that it appears multiple, but whether the multiplicity is composed of two or three separate stimuli cannot be definitely stated at this time interval. It is not till an interval between the stimuli of 27 sigma is reached that the double nature of the sensation becomes quite definite. The incidence of the first signs of reduplication occurred constantly in both observers at an interval of 10·6 sigma between the stimuli. The judgment error in either direction was not found to exceed 0·5 sigma. As in the case of summation the results were the same when obtained from the observation of the dull touch-like sensations in the forearm or the pain-like sensations from the wrist and finger.

If the two stimuli be applied by two separate electrodes placed side by side, the results obtained are identical with those from a single electrode. The appearance at this specific time interval of the sensation of reduplication does not therefore depend on the stimulation of identical nerve fibres. Further it does not depend on the stimulation of identical nerves. If one electrode stimulates the tip of the little finger (ulnar nerve) and the other the tip of the index finger (median nerve) the time relationship of the occurrence of reduplication remains unaltered, although judgment becomes more difficult for the subject. If corresponding fingers on the right and left hands be selected for successive stimulation it was found that judgment of the exact point of reduplication of the sensation became extremely difficult and it was impossible with precision to fix the reduplication point which occurred at a stimulus interval of about 25 to 30 sigma. Similar lengthening of the reduplication time was noted when the homolateral arm and leg were stimulated.

Certain inferences may be drawn from the foregoing experiments. The central nervous mechanism serving the sensation of reduplication has the same functional time relations whether both successive stimuli be applied to the same nerve fibre, to different nerve fibres, or to different but adjacent peripheral nerves. The sensory change interpreted as reduplication corresponds very closely in its stimulus interval to that at which sensory summation appears to wane. These functional time relations correspond very closely to those observed in the muscular effector organ. Fig. 2 shows that when the ulnar is stimulated the waning of muscular summation in the abductor minimi digiti occurs at about the same stimulus time interval as does the waning of sensory summation and the appearance of reduplication on the sensory side.

When a double stimulus is felt as such we are entitled to assume that we have a measurement of what must be the greatest possible duration of the refractory period of the central receptor neurones. These are successively responsible for the ultimate presentation of the sensation at the level at which judgment is pronounced, but we are by no means entitled to assume that we have also a measure of the lower limits of this period. The ultimate datum, reduplication or non-reduplication, is not a pure sensation, but a judgment involving reference to the ideas of oneness or twoness, which again in this connection probably have temporal connotations. It may well be that the whole of this complex mechanism requires for its functioning the relatively large stimulus interval of 11 sigma, whilst individual neurones in any part of the nervous system respond to stimuli at the same interval as that found for the refractory period of the lower sensory neurone.

The fact that Cooper and Denny Brown were able to show that the cortex in the cat can follow a maximum stimulation by break shocks at the rate of 180 in the second would indicate a fairly close correspondence with the refractory period determined in our experiments for the lower sensory neurone.

The diminution of the intensity of the sensation beginning at the stimulus interval of 12 sigma continues through increasing stimulus intervals, fading gradually into the intensity of the single sensation when each stimulus is felt as absolutely discrete.

The velocity of conduction in afferent nerves.—The fact that the judgment of the point of reduplication is effected with equal accuracy when the stimuli are administered to separate nerve fibres makes it possible to utilize the reduplication point for the estimation of the velocity of conduction of afferent nerves.

If we know the minimum stimulus interval at which two successive stimuli give a reduplicated response when both stimuli are administered at the same spot on the periphery of a sensory nerve, it is sufficient, in order to obtain the velocity of conduction, for the reduplication stimulus interval to be determined when the two electrodes are separated by shifting the second stimulating electrode a known distance along the course of the nerve. The velocity is obviously most easily estimated when dealing with a subcutaneous sensory nerve with straight course permitting some degree of accuracy in linear measurement. The ulnar is the best nerve fulfilling these requirements. The electrode of B circuit conveying the second stimulus is strapped on to the ulnar nerve at the olecranon. The electrode of A circuit conveying the first stimulus is

successively fixed in two positions, first over the ulnar nerve at the wrist and secondly on the palmar side of the terminal phalanx of the little finger. The length of nerve lying between these three points may be estimated with a possible error of about ±5 per cent. It has already been stated that when the same point of skin is subjected to a double stimulus, the minimum reduplication time is the same and determinable within 0·5 sigma wherever the selected point may be, and that when two points of skin are taken for the two respective stimuli the reduplication time can be determined with equal accuracy.

It was found that the reduplication time with the electrodes at the olecranon and the wrist could be determined within ±0·5 sigma. The reduplication time when the electrodes were on the olecranon and the terminal phalanx of the little finger was more difficult to determine owing probably to the qualitative difference between the finger sensation which has a painful tone, and the olecranon which has a purely tactile tone, at the current strength employed for these experiments. The constant error in the finger olecranon experiments was ±0·8 sigma.

The proximal electrode is connected with the movable key. When the two keys are struck simultaneously it is obvious that the stimulus from the distal electrode will reach the central nervous system after the stimulus from the proximal electrode by such a period of time as the impulse has taken to traverse the nerve tract lying between the two electrodes. If by shifting the movable key the proximal stimulus be progressively retarded a point will be arrived at when the stimuli are judged to be reduplicated.

Subject	Stimulus interval for reduplication		Nerve length	Velocity of conduction
G.	Both electrodes on same spot	= 0·0106 secs.	—	—
	Electrodes on wrist and olecranon	= 0·0146 ,,	30·2 cm.	75 m. per sec.
	Electrodes on terminal phalanx and olecranon	= 0·0170 ,,	46·5 cm.	72 m. per sec.
H.	Both electrodes on same spot	= 0·0106 ,,	—	—
	Electrodes on wrist and olecranon	= 0·0138 ,,	26 cm.	81 m. per sec.
	Electrodes on terminal phalanx and olecranon	= 0·0162 ,,	42·5 cm.	74 m. per sec.

The velocity assigned to the ulnar afferent nerve fibres would appear to be between 72 and 81 m. per second from the many hundreds of observations on the two subjects G and H. The possible error assessed from variations in the summation time and the difficulty of assigning an exact length to the nerve tract would appear to be about ± 5 per cent. These estimations of velocity are therefore somewhat less accurate than those which we previously published for the motor fibres of the ulnar nerve. In these the velocity of conduction in subject G was found to be 37·8 m. per second with an estimated probable error of ± 1 per cent.

The determination of the velocity of conduction of the afferent impulse in the human subject has given varying results. Attempts to estimate the velocity of sensory impulses based on reaction times cannot by their nature give results of any significance in the normal subject. The only attempt to investigate the velocity of the sensory impulse by objective methods has been that of Schäffer, who recorded the electromyogram of the electrically conditioned patellar reflex. He assigned a velocity of 54·4 m. per second to the motor nerve and 59·4 m. per second to the sensory nerve. His results cannot, however, claim serious consideration, they vary between 48 m. and 73 m. per second, the largest result being 52 per cent. greater than the smallest.

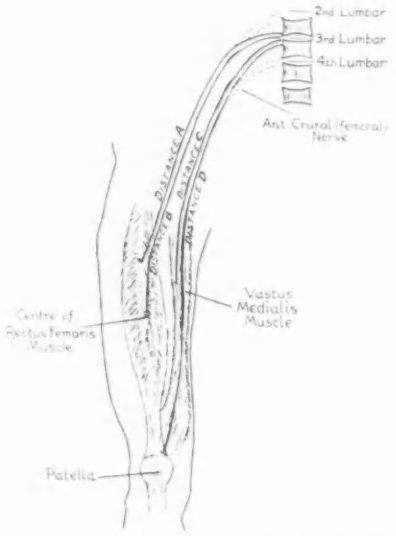

FIG. 3.—A, length of the nerve to rectus femoris to its insertion into the muscle. B, distance of A + distance between the point of insertion of the nerve and the centre of the rectus femoris muscle (midway between origin and insertion). C, length of the nerve to vastus medialis to its insertion into the muscle. D, distance of C + distance between the point of insertion of the nerve and the upper border of the patella.

If the results here recorded be valid it would appear that the velocity of the afferent nerve is about twice that of the motor nerve. Such a conclusion seems inevitable if we are to accept the velocity of 37·8 m. per second for the motor nerve and to regard the knee-jerk as a stretch reflex.

The length of nerve involved in the patella reflex has been investigated by F. A. Hoffmann, who obtained values between 110 cm. to

133 cm. in the living subject. Professor Le Gros Clark very kindly made the following series of observations for us on a series of cadavers.

The nerves were measured from the intervertebral foramen transmitting the third lumbar nerve along the trunks of the lumbar plexus to the point where the peripheral branch was inserted into the muscle. Measurements were also taken of the muscles as shown below (fig. 3).

	1 Height 5.6½	2 Height 5.11½	3 Height 5.2	4 Height 5.8½	5 Height 5.7
A	291 mm.	391 mm.	316 mm.	306 mm.	281 mm.
B	419 ,,	534 ,,	382 ,,	371 ,,	365 ,,
C	461 ,,	569 ,,	509 ,,	456 ,,	531 ,,
D	679 ,,	716 ,,	599 ,,	603 ,,	601 ,,

In the case of a man of 5 ft. 11½ in., repeated observations of the total reflex time of the knee-jerk gave values of between 24 and 26 sigma. If distance B to the middle of the rectus femoris be assigned to the efferent conduction path the time taken by an impulse travelling at the rate of 37·8 m. per second would be 14 sigma. If distance D from the patella be assigned to the afferent path the time taken by an impulse travelling at the rate of 75 m. per second would be 9 sigma; the total estimated conduction time being 23 sigma when the observed time was 24-26 sigma, thus allowing about 3 sigma as the latent period for the affector organ and no measurable time for the synapse. If the stretch receptors be placed somewhere about the middle of the quadriceps the estimated conduction time may be reduced to 21 sigma. It would appear, therefore, that assuming the absolute accuracy of the value of 37·8 m. per second for the efferent velocity, the velocity of the afferent impulse cannot be less than that deduced from the foregoing experiments.

Felix observed the reflex time of the reflex contraction of the foot muscles when the afferent nerve is stimulated at the malleolus. A large number of experiments in a series of short and tall subjects gave an average reflex time of 46 sigma for a reflex involving an estimated nerve-path of 228 cm. Apart from the impossibility of accurately measuring the nerve-length, the electromyogram is difficult to read accurately owing to the proximity of the area of stimulation to the galvanometer electrodes and the probable error is about ± 5 per cent. Felix could not by his procedure differentiate between efferent and afferent conduction and

assigns a mean velocity of conduction to the entire nerve-tract. If, however, our value of 38 m. per second for the efferent nerve be assumed to be correct, the 114 cm. efferent portion of the reflex tract will be traversed in 30 sigma, and this leaves 16 sigma for the sensory conduction time, giving a velocity of 70 m. per second for the afferent nerve. A figure which is, within the limits of error, compatible with our estimations.

That a slowing of the nervous impulse takes place towards the periphery was first suggested by Helmholtz and recent work by Felix tends to confirm this suggestion. His experiments do not, however, indicate that such difference of velocity of conduction between the central and peripheral portions of the nerve-trunk is sufficient to affect sensibly these results.

Conclusions.—A method is described for the investigation of certain functions of the sensory portion of the nervous system in the human subject.

The refractory period of various sensory nerves has been determined. The total refractory period lasted 1·7 sigma, the partial refractory period 2·3 sigma. A gradual increase of the sensory response was noted when the second stimulus fell within the partial refractory period of the first and the stimulation interval was gradually increased.

A sensation of reduplication was noted when the two stimuli were 11 sigma apart.

The sensation of reduplication was accompanied by a diminution of the intensity of sensation.

Using the method of determining the point of reduplication it was found possible to estimate the velocity of conduction of an afferent impulse in the ulnar nerve. The mean velocity of conduction was found to be 75 m. per second.

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ON THE CONNECTIONS OF THE MEDIAL CELL GROUPS OF THE THALAMUS.

BY W. E. LE GROS CLARK AND R. H. BOGGON.
(Department of Anatomy, St. Thomas's Hospital Medical School, London.)

In recent years, anatomical studies of the mammalian diencephalon have led to a much more satisfactory definition of the medially situated nuclei of the thalamus than had previously been attained. It is desirable now to follow up these morphological studies by an experimental investigation of the fibre connections of these elements. This paper is a report on a series of experiments which have been carried out with this end in view.

Methods.

The methods used in this investigation were similar to those described by us in a previous communication on the connections of the anterior nucleus (Clark and Boggon, 1933). Small electrolytic lesions were produced in the thalamus of the cat with a Clarke-Horsley stereotaxic instrument, and the course of the degenerated fibres studied by the Marchi method. Where a thalamo-cortical connection was thus found, this was corroborated in the cat and the rat by making lesions in the corresponding cortical region and studying the cellular atrophy in the thalamus which ensued after an interval of approximately three months.

For the purposes of our experiments ether anæsthesia proved the best, but it requires considerable skill to keep the animal at the correct level of anæsthesia during the whole of the operation. No abnormal bleeding was followed by its use. In our hands intraperitoneal dial was unsuitable, as a dose sufficient to keep the animal unconscious and quiet delayed the return of consciousness for an inconveniently long period.

We have found the papers by Rioch on the carnivore diencephalon (1929 and 1931), and a recent paper by Ingram, Hannett and Ranson (1932) of great value in delineating the thalamic nuclei in our sections of the cat's brain.

EXPERIMENTS.

Cat 6.—Operated April 8, killed April 26.

The right cerebellar fossa was opened and the Clarke-Horsley apparatus applied to the head. The electrode was inserted 12 mm. in front of the central point, $1\frac{1}{2}$ mm. to the left, and 5 mm. upwards (instrumental error being allowed for). A current of 5 milliampères was allowed to flow for five minutes.

Microscopical examination.—The extent and position of the electrolytic lesion may be seen by reference to fig. 1. It lies mainly to the left of the mid-line in the rostral part of the thalamus, encroaching very slightly on the right wall of the third ventricle. It involves here on the

FIG. 1.—Section through the rostral part of the thalamus of Cat 6, showing the lesion. × 4. Am, Anteromedial nucleus. Av, Anteroventral nucleus. F, Fornix. La, Lateral nucleus.

left side the periventricular system, the most medially situated fasciculi of the inferior thalamic peduncle, the most medial part of the anteromedial nucleus, and close to the mid-line the commissura interanterodorsalis. More rostrally, the lesion extends into the most medial fasciculi of the left descending fornix column and into the most medially situated fibres of the inferior thalamic peduncle of the right side.

From the site of the lesion on the left side of the mid-line, degenerated fibres extend dorsally to the stria medullaris, and ventrally in the inferior peduncle and fornix. None of the degenerated fornix fibres

run into the pre-optic region. The fibres of the interanterodorsal commissure contain scattered Marchi granules which can be followed up into the anterodorsal nucleus of either side. On the other hand the anteroventral nucleus is quite clear of osmic deposit. There is no Marchi degeneration in the internal capsule or in any part of the cerebral cortex.

Caudally at the level of the middle of the thalamus, the lesion produced by the track of the needle lies immediately to the left of the mid-line and medial to the mammillo-thalamic bundle, cutting across the periventricular system of fibres. Here a few fine fasciculi of degenerated fibres extend up in the periventricular system to the paratænial nucleus, and also into the dorsomedial nucleus, the greater part of which, on the left side, is finely sprinked with osmic granules. A few degenerated fibres can also be followed ventralwards in the periventricular system for a very short distance, but none are seen to reach down into the hypothalamus. From the site of the lesion at this level Marchi granules extend for a short distance medially and laterally in the fibres which run transversely in the region of the nucleus centralis medius.

Thus, in this specimen, the Marchi degeneration resulting from a lesion close to the mid-line of the thalamus is confined to immediately neighbouring regions, with the exception of the degenerated fibres in the fornix and the inferior peduncle of the thalamus. The distinct degeneration in the fine fibres pervading the dorsomedial nucleus provides evidence of an ascending connection between the periventricular system of fibres and this nucleus.

Cat 7.—Operated April 12, killed April 29.

In this case the electrode was inserted to a point 1·2 mm. anterior to the central point, 1½ mm. to the left of the mid-line, and 5 mm. above the horizontal plane. A current of 5 milliampères was allowed to flow for five minutes.

Microscopical examination.—The electrolytic lesion was found to be situated in the upper end of the left habenulo-peduncular tract reaching dorsally to a point 1·5 mm. below the caudal extremity of the habenular ganglion, and medially to about 1 mm. from the mid-line. It measures just under 3 mm. in vertical and 2 mm. in transverse diameter. The medial margin of the central median nucleus is involved, as well as some of the fibres of the periventricular system at this level (fig. 2). More caudally, the lesion extends into the fibres which are coursing dorsomedially into the posterior commissure, and the latter is densely

stained with the osmic acid. Rostrally, the lesion does not reach forward further than the level of the habenulo-peduncular tract.

Degenerated fibres radiate out from the site of the lesion in all directions, but none can be traced for more than a short distance (except, of course, those in Meynert's bundle). Some pass dorsally into the substance of the pretectal nucleus, laterally into the centre median nucleus and the medial margin of the lateral nucleus, and ventrolaterally into the most medial part of the ventral nucleus (nucleus ventralis, pars medialis). Others run ventrally in the periventricular system, but do not extend into the hypothalamus.

In sections passing through the middle of the thalamus rostral to the lesion, the medial medullary lamina contains scattered Marchi

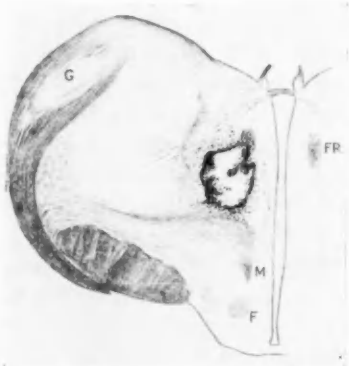

FIG. 2.—Section through the caudal part of the thalamus of Cat 7, showing the lesion. × 4. F, Fornix. FR, Fasciculus retroflexus. G, Lateral geniculate body. M, Mammillo-thalamic tract.

granules which permeate the substance of the nucleus paracentralis and nucleus centralis lateralis, and some fine degenerated fibres are seen to cross over through the nucleus centralis medius to reach the medial medullary lamina of the opposite side. It was not possible to determine with certainty to which nuclei these intralaminar fibres are specifically related.

Sections through the anterior part of the thalamus—at the level of the anterior nucleus—show no definite Marchi degeneration. In none of the sections (including those taken through the frontal lobes) were degenerated fibres found to reach the internal capsule or the cerebral cortex.

This experiment indicates that, with the possible exception of fibres

passing into the mesencephalon (for the investigation of which the lesion was not suitable), the efferent connections of the centre median nucleus are entirely intrathalamic.

Cat 19. Operated September 20, killed October 5. The electrode was inserted in the mid-line to reach 9 mm. in front of the central point and 4 mm. above the transverse plane. A current of 7 milliampères was allowed to flow for seven minutes.

Microscopical examination.—The most caudal section of the series shows the track of the needle passing through the cerebral aqueduct and involving here the most caudal and ventral fibres of the posterior

FIG. 3.—Section through the middle of the thalamus of Cat 19, showing the lesion. × 4. F, Fornix. G, Lateral geniculate body. M, Mammillo-thalamic tract.

commissure. Degenerated fibres of this commissure extend out on either side into the pretectal nucleus, the nucleus of the posterior commissure, and further ventrally into the dorsal part of the tegmentum. The habenulo-peduncular tracts are filled with Marchi granules as the result of their partial involvement by the lesion at their upper extremity. There is no osmic deposit in the cerebral peduncles. At the caudal end of the thalamus the track of the needle passes forwards in the mid-line at the upper margin on the third ventricle immediately ventral to the habenular ganglion. From the site of this small lesion very fine degenerated fibres can be traced down in the periventricular system as far as the level of the ventral medullary lamina, but not into the hypothalamus.

The electrolytic lesion lies in the middle of the thalamus at the level of sections passing through the optic chiasma and the rostral pole of the lateral geniculate body (fig. 3). It is in the mid-line, extending from the stria medullaris to the ventral margin of the nucleus centralis medius, measuring 3 mm. in vertical and 2·4 mm. in transverse diameter. As may be seen from the figure, the lesion involves on the right side practically the entire dorsomedial nucleus, as well as the medial margin of the nucleus centralis lateralis, while on the left side it involves approximately the medial half of the dorsomedial nucleus. It extends forwards to the rostral extremity of the dorsomedial nucleus, cutting into the medial olfacto-habenular tract and the commissura interanterodorsalis, but does not reach the anterior nuclei. It should be noted that nowhere does the lesion extend lateral to the medial medullary lamina.

From the site of the lesion, fascicles of degenerated fibres run through the medial medullary lamina, and ventrally in the inferior thalamic radiations mainly to the lateral side of the mammillo-thalamic tract. A fine deposit of Marchi granules is also present in the commissural fibres coursing through the nucleus centralis medius and nucleus paracentralis, reaching out to the medial part of the nucleus centralis lateralis. The degenerated fibres of the inferior thalamic radiations are more numerous on the right side and run downwards and forwards to collect at the medial extremity of the internal capsule. Further forwards, in sections passing through the caudal part of the septum, the Marchi staining of these fibres is very distinct and is localized to the fasciculi which lie immediately dorsal to the intermediate root of the olfactory tract. The grouping of the degenerated fibres in sections through the rostral part of the septum is shown in fig. 4A. It will be noted that they reach down close to the basal surface of the brain and, at this level, some of them run laterally through the extreme anterior end of the lenticular nucleus to reach the external capsule. There is no Marchi staining in the anterior commissure, nor in the caudate nucleus. In sections further forwards—through the frontal pole of the hemisphere—the degenerated fibres are seen in the most ventral part of the centrum ovale, extending up almost to the floor of the lateral ventricle. Here, also, they can be seen extending into the deeper layers of the cortex of the gyrus proreus (which are sprinkled with a very fine Marchi deposit), and reaching from the upper lip of the sulcus presylvius down to the orbital " keel " of the frontal lobe (fig. 4B). By reference to the charts of the cortical areas in Winkler and Potter's classical atlas of the cat's

brain, it is evident that these corticipetal fibres from the dorsomedial nucleus are terminally related to areæ prefontalis, orbitalis lateralis, orbitalis intermedia and orbitalis medialis. These areas form component parts of the prefrontal granular cortex.

FIG. 4A.

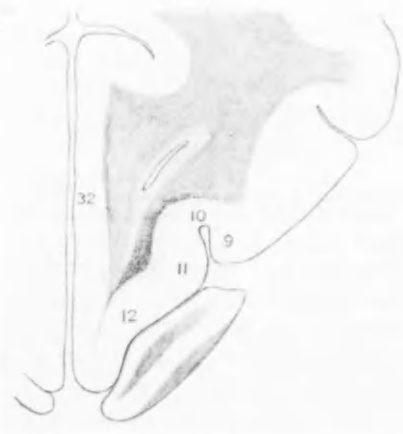

FIG. 4B.

FIG. 4.—A, Section through the rostral part of the septum of Cat 19, showing degenerated fibres in the internal capsule. × 4. B, Section through the frontal pole of the cerebral hemisphere of Cat 19. × 4. The numbers indicate the position of Brodmann's cortical areas.

Besides the degeneration already noted, the fibres of the interantero-dorsal commissure (which is involved by the rostral extremity of the

lesion in the mid-line) show an abundant deposit of Marchi granules which can be traced up into the anterodorsal nucleus of either side, and there is also Marchi degeneration in the inferior peduncle of the thalamus which results from the injury to the medial olfacto-habenular fibres. No certain evidence was found of degenerated fibres extending from the lesion into the lenticular nucleus.

Cat 21.—Operated September 27, killed October 10. The electrode was inserted up to a point 8 mm. anterior to the central point, 1 mm. to the left of the mid-line, and 4 mm. up. A lesion was made by passing a current of 7 milliampères for five minutes.

FIG. 5.—Section through the middle of the thalamus of Cat 21, showing the lesion. × 4. F, Fornix. M, Mammillo-thalamic tract.

Microscopical examination.—At the caudal margin of the thalamus the lesion lies close to the mid-line on the left side, extending almost from the upper extremity of the fasciculus retroflexus above to the ventral medullary lamina below. At this level it occupies much the same position as the lesion in Cat 7 which is illustrated in fig. 2. Further forwards it is situated rather more ventrally and, in the middle of the thalamus (in sections passing through the optic chiasma, the rostral pole of the lateral geniculate body, and the caudal end of the corpus callosum), it lies up against the mid-line in the most medial part of the ventral region of the thalamus (fig. 5). The lesion is approximately circular in section, measuring 1·5 mm. in diameter. It involves the nucleus centralis medius close to the mid-line and the nucleus sub-

medius, and more caudally the upper end of the fasciculus retroflexus, the nucleus parafascicularis, and the medial part of the centre median nucleus. The dorsomedial nucleus has escaped injury (except, possibly, for its extreme caudal pole). Two sections in front of that shown in fig. 5, the lesion is no longer present.

From the site of the lesion, Marchi degeneration extends into the ventral part of the dorsomedial nucleus, in the periventricular fibres as far up as the habenular ganglion (but not ventrally below the level of the lesion), laterally in the fibres of the commissura media into the nucleus paracentralis and nucleus centralis lateralis of both sides, and slightly in the fibres of the medial medullary lamina. It is also present in the root of the left habenulo-peduncular tract (which is involved by the lesion at the caudal end of the thalamus), and in the fasciculi of the inferior thalamic radiations which course downwards and forwards towards the ventral medullary lamina. The degenerated fibres in the latter fasciculi evidently arise in part from the dorsomedial nucleus, having been intercepted by the lesion on their way down, and, as the sections are traced rostrally, they are observed to pass into the medial end of the zona incerta and so reach the medial margin of the pes pedunculi. A few of them can be followed further forwards in the most medial fasciculi of the internal capsule (lying immediately adjacent to the intermediate root of the olfactory tract) and ultimately into the extreme rostral part of the external capsule, where they disappear in the same region of the cortex into which fibres were traced from the dorsomedial nucleus in Cat 19. The majority of the degenerated fibres which reach the ventral medullary lamina from the lesion run ventrolaterally in amongst the most medial fasciculi of the internal capsule and, having penetrated the latter, turn laterally to reach the globus pallidus, beyond which they could not be traced with certainty. There is no Marchi degeneration in the inferior peduncle of the thalamus.

Cat 22.—Operated September 30, killed October 12. The electrode was inserted to reach 8 mm. in front of the central point, 1 mm. to the left of the mid-line, and 4 mm. above the transverse plane. A current of 7 milliampères was passed for five minutes.

Microscopical examination.—The track of the needle passes forwards in the mid-line along the cerebral aqueduct, and at this level a slight injury to the ventral fibres of the posterior commissure has given rise to well-marked Marchi degeneration. The main lesion is quite small, measuring 1·7 mm. in transverse and 3 mm. in vertical diameter, and lies in the mid-line close to the dorsal surface of the thalamus. It

involves only the nucleus paraventricularis anterior (extending up as far as the ventral margin of the habenular ganglion), the extreme medial margin of the dorsomedial nuclei of both sides, and the nucleus centralis medius.

A fine Marchi deposit is present in the fibres of the commissura media extending laterally on both sides as far as the nucleus centralis lateralis and also in the fibres of the periventricular system immediately adjacent to the lesion. No degenerated fibres extend into the hypothalamus and none reach the internal capsule. There is a slight degree of Marchi degeneration in the most caudal fibres of the inferior thalamic peduncle of the left side.

Cat 18.—Operated July 22, killed October 13. Under ether anæsthesia the bone over the right frontal area was removed as far as necessary. The frontal pole of the hemisphere was divided in a transverse plane 1 cm. from the tip with a sharp tenotome. The ensuing hæmorrhage was controlled and the wound sutured. The cat made a good recovery from the operation and exhibited no unusual symptoms.

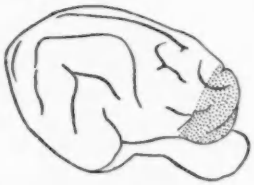

FIG. 6.—Lateral view of the right hemisphere of Cat 18, showing the extent of the frontal cortex whose connections with the thalamus had been severed by the lesion.

The extent of the lesion in the right frontal lobe was studied after death by making sections of the fresh brain and studying them with the aid of a binocular dissecting microscope. The cortical area which had been severed from the rest of the brain by the lesion is indicated by the stippled area in fig. 6. The corpus striatum was not involved by the injury.

Microscopical examination.—Complete serial sections were taken through the thalamus and stained with Borell's methylene blue. In the most rostral sections, the course of the degenerated fibres through the anterior thalamic radiations is marked out by blue staining.

The elements of the anterior nucleus and of the lateral nucleus, as well as the nucleus centralis lateralis, nucleus centralis medius, nucleus reuniens, centre median nucleus, nucleus parafascicularis, nucleus submedius and nucleus paratænialis are all normal on the side of the lesion,

and stand out in contrast with the atrophic areas in the dorsomedial and ventral nuclei.

In rostral planes, the cells of the lateral third of the dorsomedial nucleus (extending dorsally up to the habenular nucleus) have undergone almost complete atrophy. More caudally, the degeneration is a little more extensive. At its posterior extremity, the nucleus is split into medial and lateral parts by the lateral root of the habenulo-peduncular tract. The cells of the lateral part are atrophied, while those of the medial part are unchanged.

FIG. 7.—Section through the dorsal part of the thalamus of Cat 18, showing the cell atrophy which has followed on a lesion of the frontal cortex. × 20. This figure has been drawn from a section stained with Borell's methylene blue, by means of a projection apparatus. a, Area of cell atrophy in the lateral part of the nucleus dorso-medialis; Cl, nucleus centralis lateralis; Cm, nucleus centralis medius; Dm, nucleus dorso-medialis; La and Lb, nucleus lateralis; Pc, nucleus paracentralis; Sm, stria medullaris; V, nucleus ventralis, pars arcuata.

Thus, by reference to the cellular atrophy which affects it after an injury to the frontal pole of the hemisphere, the dorsomedial nucleus in the cat may be subdivided into medial and lateral portions. An examination of Nissl preparations of the normal nucleus shows that these two

parts can also be differentiated on the basis of cytoarchitecture, the cells of the lateral part (connected with the frontal cortex) being smaller and more fusiform in shape.

Atrophy of cells is also to be found very distinctly in the ventral nucleus. Most rostrally this affects, to a moderate extent, the ventromedial half of the nucleus ventralis, pars anterior. Behind this level the atrophy is mainly confined to the ventral nucleus, pars medialis, which, except at its most anterior part, is almost completely degenerated, very few normal cells remaining. On the other hand the nucleus submedius (? = nucleus medialis ventralis), which lies medial to the mammillothalamic tract, appears normal. At the more anterior levels of the thalamus the pars externa of the ventral nucleus also shows an area of cellular atrophy, but caudally this is only slightly indicated. At caudal levels the cellular degeneration of the ventral nucleus involves the medial part of the pars arcuata, extending out 3·5 mm. from the mid-line. This atrophy shrinks in more posterior sections until it is limited to a small circumscribed patch in the medial " tail " of the arcuate nucleus.

The nucleus paracentralis is normal at rostral levels, but posteriorly many of its cells have undergone complete degeneration, so that the area of cellular atrophy in the medial part of the ventral nucleus becomes here linked up with that in the lateral part of the dorsomedial nucleus. No cellular atrophy is found behind the level of the thalamus.

Experimental lesions in the rat's brain.—One of us has already adduced evidence, on the basis of the Marchi method, of a reciprocal fibre connection between the dorsomedial nucleus and the frontal pole of the rat's brain (Clark, 1932 *b*). In the following three experiments on rats, a small lesion was made in the tip of the frontal pole of the left hemisphere with a discission needle. The animals were killed approximately three months later and the extent of the lesion studied in the fresh brain with the aid of a binocular dissecting microscope. A complete set of serial sections through the thalamus was then prepared and stained with Borell's methylene blue.

Rat FF. 1.—Examination of the brain after death showed that the lesion had severed the rostral 3 mm. of the frontal pole from the rest of the hemisphere and involved the olfactory peduncle. The microscopical sections show that the anterior end of the striatum was also injured in front of the anterior commissure.

Serial sections through the thalamus demonstrate the following features. On the side of the lesion there is very distinct atrophy (indicated by marked shrinkage and poor staining of the cells) of the

lateral third of the dorsomedial nucleus and also of the whole of the nucleus medialis ventralis. On the other hand the nucleus centralis medius, nucleus paracentralis, nucleus centralis lateralis, and more caudally the nucleus parafascicularis and its lateral extension (the homologue of the centre median nucleus) are quite normal in appearance. Similarly, there is no sign of cellular atrophy in the anterior, ventral, lateral, or other thalamic nuclei. The degenerated areas are very clearly defined and stand out in conspicuous contrast with the rest of the thalamus in methylene blue sections.

Rat FF. 2.—Examination of the brain after death showed that the lesion had cut transversely across the left frontal pole 4·5 mm. behind its rostral extremity, and had involved the olfactory peduncle and the anterior 1·5 mm. of the striatum (caudate nucleus + putamen).

Serial sections through the thalamus demonstrate an almost complete atrophy on the left side of the lateral two-thirds of the dorsomedial nucleus, of the nucleus anteromedialis, nucleus medialis ventralis, and the left half of the nucleus reuniens. In contrast with these degenerated areas there is no apparent change in the following nuclei: anterodorsal and anteroventral, paratænial, lateral, ventral, paracentral, lateral central, nuclei of the mid-line (except the left half of the nucleus reuniens), parafascicular and centre median.

Rat FF. 3.—In this case the lesion was extremely small. Besides injuring the olfactory bulb, it was confined to the extreme tip of the frontal pole, just incising the thickness of the cortex at this point.

Serial sections through the thalamus showed no cellular atrophy in any of its elements.

Discussion and Summary.

The discussion of the results of the experiments which have been recorded in this paper may best be accomplished by considering separately the various nuclei involved.

Nucleus dorsomedialis.—(Nucleus medialis *a*, nucleus medialis dorsalis of other authors). In Cat 19, the lesion was practically confined to this nucleus and the immediately adjacent mid-line nuclei. Marchi staining showed that, in addition to sending fibres into the medial medullary lamina and the periventricular system, the nucleus emits corticipetal fibres which run ventrally into the most medial fasciculi of the internal capsule, and then pass forwards in close relation to the septum and the basal surface of the brain, to terminate in the granular cortex of the frontal lobe represented by areæ prefrontalis, orbitalis lateralis, intermedia and medialis. Conversely, in Cat 18 and in Rats

FF 1 and FF 2, a very distinct cellular atrophy affecting the lateral part of the dorsomedial nucleus was found after severing the connection between the frontal pole of the hemisphere and the thalamus. It may therefore be concluded that cortical projection fibres from the dorsomedial nucleus are distributed to the prefrontal region of the cortex, and this confirms the results obtained by the Marchi method in the rat's brain (Clark, 1932 *b*). It is relevant to recall that in the higher primates this region of the cortex undergoes a remarkable expansion, and that, coincidently with this, the dorsomedial nucleus in these forms shows a progressive enlargement and a clear differentiation into lateral and medial elements (Friedemann, 1912; Pines, 1927; Clark, 1930). It may be inferred from our experiments that the lateral element is the source of the corticipetal fibres. We believe that the connections of the dorsomedial nucleus with the periventricular system on the one hand, and with the prefrontal region of the cortex on the other, may be of considerable significance. It is evident that the nucleus provides a mechanism through which the cortical levels can be brought into direct functional relation with the activities of the hypothalamus.

Nucleus submedius.—This nucleus in the cat's thalamus is probably homologous—at least in part—with the nucleus medialis ventralis of the rat. In Cat 21 the nucleus submedius was destroyed by a small localized lesion. As a result, in addition to a small amount of degeneration in the periventricular system, degenerated fibres were found passing ventrally to the medial part of the internal capsule. A very few were traced forwards to the frontal pole of the hemisphere, but we think it certain that these originate in the dorsomedial nucleus, having been injured by the lesion on their way down past the nucleus submedius. Most of the degenerated fibres, which evidently have their origin in the submedial nucleus, penetrate the internal capsule to reach the lenticular nucleus. This connection corresponds precisely with that of the nucleus medialis ventralis in the rat's brain as demonstrated by the Marchi method (Clark, 1932 *b*). A marked atrophy of the whole of this nucleus was found in Rats FF 1 and FF 2, following a lesion in the frontal pole of the hemisphere, and the significance of this requires further investigation. In both these cases, however, the rostral part of the striatum (caudate nucleus + putamen) had been involved by the injury. This fact, together with the absence of positive evidence based on Marchi degeneration, precludes the assumption of a direct cortical connection of the nucleus medialis ventralis.

Nucleus anteromedialis.—In one experiment (Cat 6) this nucleus

was involved by the lesion, but only to a very slight extent at its medial margin. In Rat FF 2, the whole nucleus had undergone atrophy following a lesion of the frontal pole of the hemisphere. As in the case of the nucleus medialis ventralis, further experimental inquiry is needed to elucidate the meaning of this degeneration (e.g., whether it is secondary to an injury of the cortex involving that part of the area cingularis which extends on to the medial surface of the frontal lobe, or whether it is associated with the injury of the rostral end of the striatum). In this experiment on the rat, the sharp contrast between the degenerated anteromedial nucleus and the unaffected anteroventral nucleus may be noted, for it serves to emphasize the distinction between these two elements which is apparent in their normal cytological features.

Nucleus centralis lateralis.—This nucleus remained unaffected by lesions of the frontal pole of the hemisphere. Marchi sections indicate that it is closely related to the commissural fibres of the thalamus which run through the nucleus centralis medius, and that, by way of its fibre connections with the latter nucleus, it may also serve as a link through which the more laterally situated nuclei of the thalamus are brought into relation with the mid-line nuclei and the periventricular system of fibres.

Centre median nucleus.—This nucleus was partly destroyed in Cat 7. No degenerated fibres were traced from the site of lesion beyond the immediately adjacent thalamic nuclei (except in the habenulo-peduncular tract which was also injured in this experiment). This result is in accord with the experiment recorded elsewhere in which the centre median nucleus was also injured (Clark and Boggon, 1933). It may also be noted that lesions in the frontal pole of the hemisphere led to no atrophy in the nucleus. Thus it appears that the centre median nucleus is an intrathalamic mechanism concerned rather with the linking up of other thalamic elements, a conclusion reached by one of us on the basis of comparative anatomical studies (Clark, 1932 *a*).

Nuclei of the mid-line.—Following lesions limited practically to the mid-line nuclei (nucleus paraventricularis anterior, nucleus commissuralis interanteroventralis, nucleus commissuralis interanterodorsalis, nucleus centralis medius), degeneration was limited to commissural fibres and periventricular fibres. The former could be traced out either to the anterodorsal nucleus, or through the nucleus paracentralis as far as the nucleus centralis lateralis. They could not with certainty be followed further. The degenerated periventricular fibres were followed up dorsally into the paratænial nucleus and the dorsomedial nucleus.

Ventrally they could be traced in a subependymal position as far as the junction of the thalamus proper with the hypothalamus, but in no case actually into the substance of the hypothalamus. Rioch (1931) has demonstrated with Weigert material a fibre connection between the mid-line nuclei of the thalamus and the hypothalamic nuclei. From our experiments, it appears certain that (so far as myelinated fibres are concerned) this connection is established by short relays of neurones.

Thalamo-cortical projection fibres.—The positive evidence which we have adduced of cortical connections of the dorsomedial nucleus and, in a previous communication, of the anterior nucleus (Clark and Boggon, 1933), indicates that, at least in lower mammals, the prefrontal cortex and the cortex of the gyrus cinguli must be included among the thalamocortical projection zones (in addition to the somato-sensory, visual and auditory areas). This result finds no support in the conclusions attained by Poliak in his recent elaborate monograph on the afferent fibre systems of the cerebral cortex in primates (1932). It should however be noted that: (1) this author did find Marchi degeneration in the gyrus cinguli along the dorsal surface of the corpus callosum in one of his experiments (No. 2) after a lesion involving the anterior limb of the internal capsule, but he does not appear to consider the question of this degeneration being the result of an interruption of thalamo-cortical fibres; (2) in none of his experimental lesions were the fibres of the anterior thalamic radiations, which course forwards close to the basal surface of the brain and which (in the cat and the rat) contain the projection fibres from the dorsomedial nucleus, interrupted by the injury.

We are much indebted to the British Association for the loan of the Clarke-Horsley stereotaxic instrument which we used in the course of the experiments recorded in this paper.

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ON A CORTICAL FLEXOR TONE IN THE FORE-LIMB OF THE CAT, WITH OBSERVATIONS ON THE HEMIPLEGIC ATTITUDE IN MAN.

BY D. M. BLAIR AND R. J. S. McDOWALL.

(From the Departments of Anatomy and Physiology, King's College, London.)

VARIOUS clinical conditions in man associated with tonic spasm have been studied from time to time in relation to the classical picture of experimental decerebrate rigidity in animals. In particular, attempts have been made to relate the postural effects seen in clinical cases of hemiplegia and paraplegia to the position assumed in animal decerebrate rigidity; and in comparing and contrasting the human and animal pictures attention has been drawn to one striking difference, that while in the animal the fore-limb shares with the hind-limb a position of rigid extension, in man the arm is usually in a position of rigid flexion and adduction. Recent papers by Manson and Ferguson (1930) and by Brain (1927), for example, emphasize this difference and contain brief reviews of earlier discussions of the clinical counterpart of animal decerebrate rigidity.

The following observations seem to have a direct bearing on this problem of difference. They are put forward with all due reserve in that they are observations made on animals under general anæsthesia. But it may be claimed with some justice that phenomena seen under successive stages of general anæsthesia are not unconnected with the effects produced by experimental or pathological lesions of the brain. For, in general, the successive stages of anæsthesia reflect effects on successive brain levels. And, as will be seen, our observations are supported by experimental results and throw a suggestive light on pathological conditions. Careful observation of the behaviour of both animals and man under anæsthesia is indeed likely to provide a fruitful field for investigation of cerebral function.

In cats under avertin anæsthesia we have repeatedly noted a stage at which the hind-limbs are rigidly extended while the fore-limbs are rigidly flexed and adducted. As anæsthesia deepens beyond this stage, a position is assumed of extensor rigidity in all limbs, which is succeeded in turn by a stage of complete relaxation of all limbs comparable to

what is termed "surgical anæsthesia" in human operative practice. These stages have been observed to occur in reverse order when an animal is recovering from deeper anæsthesia. The stage of flexed adducted fore-limbs with extended hind-limbs has been noted most regularly when avertin has been given intravenously during preliminary ether anæsthesia. In one consecutive series of eight cats anæsthetized in this way the stage was seen distinctly in six, indefinitely in one, and not at all in another. In the last case a considerable amount of ether was given before giving avertin. This particular stage has been observed much more irregularly when ether alone, or avertin alone, was employed.

At the stage referred to, the animal is completely unconscious; while recovering from it no signs of struggling are seen until a much lighter stage of anæsthesia is reached. The position of the limbs is entirely unchanged by altering the position of the animal as a whole, or of its head and neck in relation to its trunk.

As this position preceded, in deepening anæsthesia, the stage of quadrupedal extension resembling the attitude of decerebrate rigidity, it seemed likely that it was related to supra-mesencephalic function. We therefore investigated the effect on this posture of removal of cerebral cortex. It was found that a suitable removal of cortex on one side caused the flexion in the opposite fore-limb to be replaced by extension. It was more difficult to observe on the trephined animal the typical stage of rigidly flexed fore-limbs and rigidly extended hind-limbs, because the deep anæsthesia induced before trephining was not always promptly recovered from. The amount of avertin by then given in some cases was apt to induce a delayed effect going on to the production of complete muscular relaxation, lasting, it might be, for many hours. The blood-pressure in such cases remained normal, so that this condition was not one of surgical shock, but rather an intense "sleep" condition, such as has been known to occur at times in the clinical use of avertin. But, nevertheless, in some animals we were able to observe, after trephining, the position with rigidly flexed fore-limbs and rigidly extended hind-limbs; unilateral removal of cortex was then followed by extensor rigidity in the opposite fore-limb. In some other cases where, after trephining, both fore-limbs persisted in a condition of only slight rigidity and partial flexion, and on removal of cortex on one side little or no change in the attitude of the limbs took place, the fore-limb on the side of cortical removal was found to resist passive extension slightly or even to respond with a definite flexor pull, while the fore-limb, the cortical connections

of which had been interfered with, now showed no such signs of flexor tone.

Thus we found that this tonic flexor position of the fore-limb which is "released" at a certain stage of anæsthesia, is abolished by contralateral removal of cerebral cortex, just as we had previously noted that it disappeared with deepening anæsthesia. It therefore seems to be maintained by cortical influence.

An attempt was made to determine more precisely whether the maintenance of this flexor tone was associated with any particular area or extent of cortex upon ablation of which the flexor tone disappeared. Removal of cortex was done on one side only, also first on one side and then on the other side. Always purely contralateral effects were noted. The motor area was identified by electrical stimulation. Suitable precautions were taken to prevent drying of exposed cortex.

When removal of cortex was strictly confined to the motor area, no alteration in the incidence of this fore-limb flexor tone was observed to follow. Nor was any change seen when cortex was removed behind the motor area, on the lateral aspect of the hemisphere, and the motor area was left intact. But removal of the motor area plus an extent of cortex posterior to it rather greater in size than the motor area itself brought about a loss of flexor tone previously present in the opposite fore-limb, or caused a more definite condition of rigid flexion and adduction previously present to be replaced by rigid extension.

Removal of cortex anterior to the motor area produced no appreciable result until part at least of the motor area also was removed; then the most striking result was increased extensor rigidity of both fore-limb and hind-limb on the opposite side, as has already been described by Warner and Olmstead (1923) to take place on extirpation of the frontal cortex. These authors do not mention any difference of effect in the fore- and hind-limb; we observed that while stimulation of the cut surface of the frontal region diminished distinctly the rigidity of the contralateral hind-limb, a slight flexor tone appeared in the contralateral fore-limb.

According to our observations, therefore, the maintenance of the flexor tone in the fore-limbs does not seem to be localized in any particular part of the cortex by itself. The parietal area seems to be specially concerned, but a fairly extensive ablation of cortex in this region does not bring about loss of the flexor-tone unless the motor area also is destroyed. Removal of the motor area alone does not affect the flexor tone, but we have never seen this tone disappear upon removal of any

other area of cortex while the motor area remained intact. Loss of the flexor tone seems to follow upon cutting off the motor area plus a considerable extent of adjoining cortex.

How far this flexor rigidity which we have studied in the fore-limb of the cat corresponds to the clinical flexor rigidity of the arm in man can be at present a matter for conjecture only. Rigid flexion of the arms and extension of the legs is often seen in early stages of general anæsthesia in man. Sherrington has stated, in a personal communication referred to by Brain (1927), that in monkeys prior to deep anæsthesia, there is frequently a long-maintained flexor position of the fore-limbs. It may quite well be the case that in man, owing to the high degree of integration throughout the cerebral cortex, cutting off a localized part of the cortex by a pathological lesion (as the motor area in hemiplegia) may so interfere with cortical function as a whole as to "release" a flexor tone in the arm, just as is done in incomplete anæsthesia. We have demonstrated that in the cat a cortical fore-limb flexor tone can persist after ablation of the motor area. May it be that the extensor rigidity of the arms, more rarely seen in patients with cerebral lesions, indicates a more extensive removal of cortical control than that of the motor area only, as is the case in the cat?

But the main significance of our present observations, in relation to the problem of flexor rigidity of the arm in man, is the demonstration that in the cat extensor rigidity of the hind-limbs may co-exist with either extensor *or* flexor rigidity of the fore-limbs, under different conditions. It is therefore unnecessary to force a strict comparison between the complete "decerebrate rigidity" posture in animals, and the human clinical posture of flexed arms with extended legs. If flexor rigidity and extensor rigidity are both found in the fore-limbs in animals, it is beside the point to compare the human flexor position with the animal extensor position only, and to seek an explanation of the difference in posture in the altered normal use of the fore-limbs in man.

Brain (1927) has suggested that the hemiplegic position with flexed arm and extended leg represents a "release" of the primitive anthropoid erect attitude. But it is surely unsound to argue that pathological interference with normal function of the human brain may "release" an atavistic attitude which in a lower animal is the outcome of the activity of a normally acting brain—to argue in other words, that a localized pathological lesion has the effect of putting back the hands of the evolutionary clock. The work of Richter and Bartemeier (1926) is a striking demonstration against this argument: they showed that the

decerebrate attitude in sloths reproduces in detail the upside-down hanging posture peculiarly characteristic of these animals themselves. Brain's demonstration that, when the body of a hemiplegic patient is brought into the quadrupedal-standing position, the flexed arm then becomes extended, is merely a demonstration of the activity of proprioceptive and labyrinthine reflexes, which he admits must be brought into play in order to produce this phenomenon (extension of the arm does not follow mere passive flexion of the leg upon the trunk).

Our investigation lends some support to the suggestion of Kinnier Wilson (1920) that the flexed arm position in man is an incomplete form of the decerebrate posture in animals. On the basis of our observations in the cat, we would put it that the flexor position of the arm is the result of incomplete decerebration and depends on some remaining cortical influence.

Summary.

(1) A fore-limb flexor position seen in cats under avertin anæsthesia is described.

(2) Evidence is given of the cortical nature of this flexor tone.

(3) The bearing of these observations on the human hemiplegic posture is discussed.

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Notices of Recent Publications.

The Sign of Babinski. A Study of Cortical Dominance in Primates. By J. F. Fulton and A. D. Keller. London: Baillière, Tindall and Cox. 1932. Pp. 165. Price 31s.

Physiological analysis of the Babinski response has shown it to be but an integral part of a flexion reflex of the human lower limb, which in turn represents the nociceptive flexion reflex of the decerebrate and spinal cat described by Sherrington. However, the identification has lacked completeness, since no very definite dorsiflexion of the digits seems to occur in this reflex as seen in these animal preparations.

The investigations so fully set forth in this monograph have attempted to fill this gap by a series of comparative studies of the normal and pathological plantar responses in primates. For this purpose the authors have had at their disposal a continuously ascending series of animals: macacus, baboon, gibbon and chimpanzee.

In macacus they have performed spinal cord transection and hemisection, and ablation of one or both cortical "leg areas." In the other members of the series, with the single exception of a spinal cord transection in a chimpanzee, they have confined their experiments to cortical ablations.

In all their animals in a normal state, the plantar response was found to be either absent or of the human flexor type. No dorsiflexion response of the hallux was observed. After spinal cord transection in *macacus*, the only plantar response seen was plantar flexion of the four lateral toes, but thoracic hemisection of the cord was followed by the appearance of a homolateral Babinski plantar response. Ablation of the cortical representation of the leg failed to effect a change in the normal plantar response. Similar results were obtained in the *baboon*, with the exception that ablation of the cortical "leg area" was followed on plantar stimulation by dorsiflexion and fanning of the four lateral toes, the hallux remaining inactive.

In a *gibbon*, ablation of one cortical "leg area" was followed by the appearance on stimulation of a typical Babinski response in the contralateral foot. In the *chimpanzee*, ablation of the "leg area" is followed by the appearance of a Babinski response, which consists of dorsiflexion of the four lateral toes, dorsiflexion and marked abduction of the hallux, and finally fanning and lateral deviation of the four lateral toes. Associated limb flexion is present in the earlier stages, but tends to diminish and to disappear after some weeks. The focus of the receptive field seems to be the skin of the plantar

surface of the toes, but the whole surface of the foot may be effective in favourable cases. The adequate stimulus is pinching and pricking of the skin and pressure over the region of the malleoli.

In addition to their observations on this reflex, the authors found that as the animal scale is ascended, the degree of paralysis of the affected leg after cortical ablation increases, and the extent of restoration of power diminished. Thus the dog can walk a few hours after ablation of its motor cortex, but in the chimpanzee there is profound paralysis of the affected leg and slow and imperfect recovery. This they attribute to increasing cortical dominance in the higher animals.

The authors have been fortunate in their opportunity to study this series of primates in this way, which made it possible for them to subject the flexion reflex and its digital components, and also the associated crossed plantar response, to a careful analysis. More might have been made of this opportunity. Thus, they might have mapped out the receptive field, as it has been mapped in the cat and in the human subject; they could have determined the rôle played by stimulation of deep structures, the range of adequate stimuli and the full field of motor response.

With the results actually obtained, it is reasonable for the clinical neurologist to feel some disappointment. By adopting an unsound clinical basis of study, namely, by studying the reflex in terms of Oppenheim's, Gordon's and Chaddock's signs, the study of the receptive field and of the stimulus have been confused, and from the medley of observations obtained no determination of receptive field or of range of stimuli has been possible. The limb flexion component of the response has not been adequately studied and the crossed plantar response has escaped investigation altogether. In the case of the apes we still lack, therefore, that finished analysis of the flexion reflex and its associated plantar response that we have long possessed in the case of animals so widely separated as the cat and man.

The clinician will be struck by the statement in the opening pages of the monograph that no previous physiological analysis of the Babinski response has been attempted. It appears, however, that the authors intend to convey something wholly different; namely, that no previous experimental study of the response in apes has been attempted. After all, the analysis of a reflex reaction occurring in man can be made only by the study of man, and the investigation of a simian reflex—however precise the experimental method of study—can do no more than throw light upon what happens in the ape.

Actually, human physiological studies of the Babinski response have been made and the references thereto may be found in the author's bibliography. The monograph contains a wealth of precise experimental detail and numerous illustrations mostly bearing upon points of technique. These will be of value to workers engaged in this field, and it is probably for them that the book will have its chief interest and importance.

Atlas Anatomicum Cerebri Humani. By G. JELGERSMA. Scheltema and Holkema's Boekhandel en Uitgevers Mij. N.V. Amsterdam. 1932.

This atlas consists of 108 loose plates, 43 by 32 cm. in size, of photographs of 168 sections of the human brain and spinal cord stained by the Weigert-Pal method. The first 67 are in the coronal plane, then come 7 of sagittal sections and the remaining 31 show sections in the horizontal plane. The last three plates are of horizontal sections of each segment of the spinal cord. These are the least satisfactory part of the atlas, and compare unfavourably with Bruce's atlas of the spinal cord. The sections photographed are not always truly representative of the segment. For example, C 5 has as narrow a ventral horn as C4, and D12 as D11. The sections, that is to say, are taken through the uppermost end of each segment and not through its centre. Further, the characteristic appearances of the first three sacral segments are not illustrated, only two sacral sections from low in the conus being reproduced. Apart from these three plates the illustrations are excellent and are of great value both for teaching and research. The technical skill both of the histological preparations and of their reproduction by helio is of a high order.

No descriptive text, apart from a short note on the technique employed, has so far been published with the atlas.

Die Multiple Sklerose Einst und Jetz. Von R. BING. S. 41. Basel: Benno Schwabe. 1932. Price 2 fr. (Swiss).

This reprint from the *Schweizerische Medizinische Wochenschrift* of a lecture on the nature and pathogenesis of Disseminated Sclerosis will prove useful to those unable to study the enormous literature on this disease. The author reviews shortly its history and the many conceptions of its pathology put forward by earlier and recent writers; he accepts, as most authorities do now, the view that the lesions are primarily inflammatory and the result of some exogenous cause, as an infection, but he emphasizes the part possibly played by endogenous factors. The most important contribution to our knowledge which Bing presents is the result of the careful investigation undertaken by the Public Health Authorities and the Swiss Neurological Society on the distribution of the disease in Switzerland; this brings out many facts on the possible influence of climate, altitude, occupation and social standing, as well as on the age of onset and familial incidence, which cannot be neglected in any future study of the disease.

Syndromes acromégaliques au Cours de Tumeurs intracraniennes non hypophysaires. Par P. PUECH. Pp. 39. Paris: C. Serre. 1933.

Two cases of meningiomas of the lesser wing of the sphenoid are described in patients with symptoms of acromegaly, but it must be admitted that in neither were the latter pronounced. The patients died after operations and in

their pituitary bodies small acidophil adenomata were found. In one case the pituitary was compressed by a prolongation of the tumour, but in the other it was not affected. Reference is also made to a case of glioma of the chiasma (surviving thirteen years after the onset), in which also signs of acromegaly were present. The association of acromegaly with tumours which do not involve the hypophysis is not regarded as a coincidence, but no adequate explanation of the association is offered. The literature on the subject is not adequately examined and the monograph is worthy of note only for the records it contains.

Papers relating to the Pituitary Body, Hypothalamus and Parasympathetic Nervous System. By HARVEY CUSHING. Pp. 234. Springfield and Baltimore: C. C. Thomas. 1932. Price 5 dollars.

Four separate papers dealing with disorders of the pituitary body and allied conditions which have been already published during the last three years, have been reprinted by the author in this volume. Few changes of importance have been made in the text, but the inclusion of further cases of basophil adenomas, and the addition of new observations to the lecture on the posterior pituitary hormone and the parasympathetic apparatus, help to complete the original articles.

As some of these papers have been published in journals not easily accessible, their collection into this volume is welcome and increases our debt to their distinguished author.

Le Traitement actuel de la Paralysie générale. Par P. MOLLARET. Paris: J. B. Baillière et fils. Pp. 68. 1932. Price 8 fr.

The various methods for which good results have been claimed in the treatment of general paralysis are discussed in this short volume, but in practice two only are considered nowadays, infection with malaria and intramuscular injections of stovarsol and malaria have given the best results and should be preferred.

The author adds nothing new to our knowledge of the treatment of general paralysis, but his descriptions of these methods may be useful to those inexperienced in their use.

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Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

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